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STRICTURES OF THE COMMON AND HEPATIC BILE DUCTS[1]

POSTOPERATIVE PROGRESS IN SEVENTEEN CASES

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CONTRACTURE and stricture of the common and hepatic bile ducts, producing at first intermittent obstruction but later more complete obstruction of the common bile duct, form an impressive group of cases. That most of such strictures are due to injuries to the ducts needs no further proof, and reports that just as extensive contractures of the ducts have occurred spontaneously as a result of infection either in the biliary passages or their adjacent structures without the patient having been previously operated on can be found in the literature. The possibility of a spontaneous biliary stricture being carcinomatous is illustrated by Elting's case reported by Riggs in which 3 months after the excision of a stricture at the lower end of the common bile duct and choledochoduodenostomy jaundice again appeared. A mass in the region of the choledochoduodenostomy proved to be carcinomatous. Re-examination of the specimen of the stricture previously removed revealed, after painstaking and careful search, the presence of malignant cells. Rolleston believed that Andral's case of inflammatory stricture of the common bile duct reported in 1831 was probably carcinomatous. A perforated duodenal ulcer, strangling the duct by the products of inflammation, was reported in 1876 by Morgan who also stated that in 1860 Holmes presented before a pathological society in London a stricture supposed to be due to the passage of a stone from the common bile duct. If such did occur, the possibility of the stone "fretting away at the walls of the duct" producing ulceration seems more likely to have been the cause rather than the passage of the stone. Bristowe reported a case of stricture of the intestinal portion of the common bile duct which he believed might have been syphilitic, substantiating this by demonstrating extensive small round cell infiltration surrounding the bile ducts. Two cases of fibro-adenoma in the stump of the cystic duct producing typical symptoms of common-duct obstruction were reported by W. J. Mayo in 1916.

Prior to 1914 strictures of the common or hepatic ducts were reported for the most part as single cases. In this year Jacobson reported one case of his own and reviewed 34 others from the literature. He directed attention to the various methods used in the repair of the stricture as well as to the immediate postoperative result. Ellsworth Eliot, Jr., in 1918, reported 3 cases of stricture of the hepatic and common bile ducts in which he operated. He also made an exhaustive review of the literature and grouped the cases according to the method of treating the stricture; the results in each case were recorded.

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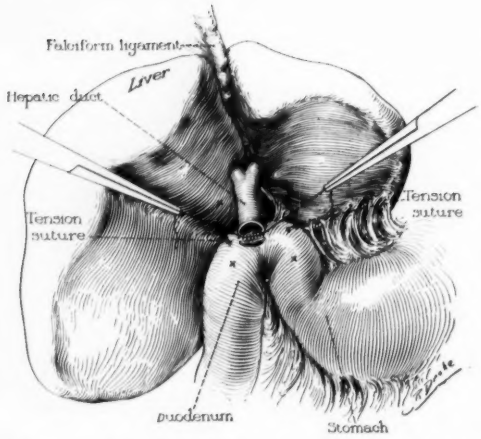

Fig. 1. Choledochoduodenostomy with end-to-side anastomosis.

McArthur, in 1923, and Douglas, in 1926, reported several of their own cases. Judd, in 1925, reported the results of operation in 48 cases of stricture of the common and hepatic ducts in which operation had been performed.

PATHOLOGIC ANATOMY

In an address on the "Functions of the Biliary Passages in Relation to Their Pathology," Wilkie, stressed the necessity of studies of the normal structures and function of the parts concerned, which require a knowledge of minute anatomy and physiology. During the last 3 years Burden, and Counseller and McIndoe have made interesting contributions to the knowledge of the structure, size, and condition of the biliary tract in health and in disease. In a study of the pathological anatomy of the bile ducts, Burden summarizes as follows:

The hepatic, cystic (distal part), and common bile ducts are identical in structure. They are lined by a layer of tall columnar epithelium which covers a surface made uneven by numerous shallow depressions. The epithelium rests directly on a thick compact layer of elastic connective tissue which makes up most of the thickness of the wall and on which the tensile strength of the duct is mainly dependent. The outside coat of the duct is composed of a loose layer of areolar tissue in which are found bundles of unstriped muscle, blood vessels, and lymphatics.

The walls of the ducts are richly supplied with glands which are situated for the most part in the outer coat, but the ducts of the glands, coming together from all directions, finally empty into ampulla-like openings which are arranged in a regular manner around the duct and communicate with its lumen. There is no evidence of true parietal sacculi or diverticula.

The ducts are provided with well developed musculature which is composed of isolated longitudinal and circular bundles situated in the outer layer of the duct and separated from each other by connective tissue. The muscle does not form a compact layer but is arranged as a loose network.

The most frequent pathologic changes in the ducts are those of inflammation. Cholecystitis is nearly always accompanied by infection in the walls of the ducts. The lesions are those of the usual chronic inflammatory type, characterized by lymphocytic infiltration and the production of fibrous tissue. The glands may retain infection and aid in its dissemination through the duct. The glands respond to the irritation by an overproduction of mucus and become dilated and cystic. The process of repair is attended by the formation of fibrous tissue which results in a thick and inelastic tube.

Judd and Counseller studied the structure of the intrahepatic biliary tree by the celloidin injection corrosion method, combined with microscopic examination of the biliary tree itself, and called attention to the fact that general obliterative cholangitis may exist months before signs of stricture. They noted that these strictures of the common bile duct differ from those following simple aseptic ligation in that the infective process was already resident in the ducts previous to the operation at which the injury was inflicted. Hence, the retained bile is rapidly infected and exacerbation of acute cholangitis follows. Although moderate dilatation usually occurs it is rarely extensive and may be absent altogether.

It must be concluded that infection of the walls of the common and hepatic bile ducts is, among other factors, causative in the production of benign strictures of these ducts. Continuation of this infection with infection of the intrahepatic branches of the biliary tree in the proportion in which they exist when compared to the normal, may determine the prognosis in each instance after operative restoration of continuity of the biliary tract. This, I believe, should be given careful consideration in studying results of the surgical treatment of

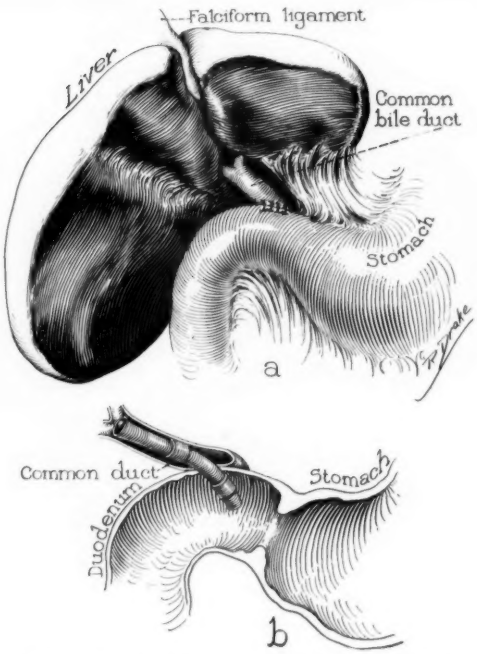

Fig. 2. Use of rubber tube in lateral choledochoduodenostomy. Case 3.

strictures of the common and hepatic ducts, remembering that if sufficient normal duct remains proximal to the stricture to permit accurate anastomosis to an incision in the duodenum, excellent results can be expected, but under different circumstances one must be content to secure improvement of health, even though short periods of jaundice and possibly of fever occur at infrequent intervals. Excellent results are always to be sought for but it is not to be expected that they can be obtained in every case.

RESULTS OF OPERATION FOR STRICTURE

In a series of 83 cases in which I operated for obstructive lesions of the common and hepatic ducts and tumors in the head of the pancreas, during the last 4 years, there were 17 in which benign stricture of the common or hepatic duct was the cause of the biliary obstruction. Fourteen of these patients are living. Seven have had excellent results, and have been free of pain, jaundice, chills and

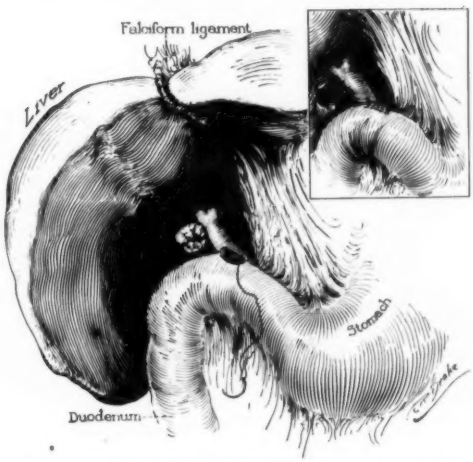

Fig. 3. Choledochoduodenostomy in a case in which operation was followed by temporary duodenal fistula. Case 1.

fever, or itching. The remaining seven have had fairly good results; they are working, and free of constant jaundice yet at intervals have a temporary incomplete biliary obstruction, as evidenced by slight jaundice, or chills and fever. Two patients died in the hospital following operation (Cases 12 and 17, tabulation). Both were deeply jaundiced at the time of operation and with serum bilirubins of 12.8 and 10 milligrams. One of these (Case 12) had a greatly enlarged liver and splenomegalia. At postmortem examination suppurative cholangitis, hydrohepatosis, and intra-abdominal hæmorrhage were found. Of further interest is the fact that the biliary obstruction had existed for 11 months before the plastic reconstruction of the stricture. The other patient (Case 17) had lost a great deal of weight, weighed 80 pounds at the time of operation, and had been deeply jaundiced for 2 months, with serum bilirubin of 10 milligrams. She had been operated on twice elsewhere for a perforated duodenal ulcer in April, 1927, and again in November, 1927, at which time the gall bladder was removed. Another patient, a woman aged 64 years, died after she had recovered from the operation. Hepaticoduodenostomy was performed, at which time only a fringe of hepatic duct remained for anastomosis to the duodenum. She left the

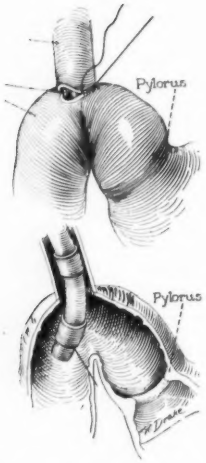

Fig. 4. Use of rubber tube in choledochoduodenostomy with end-to-side anastomosis. Case 2.

hospital 28 days after the operation, in good condition, free of jaundice, but she died suddenly 20 months later, cause unknown.

METHODS OF RECONSTRUCTION

Jacobson, Eliot, and Judd summarized the various methods employed in the restoration of biliary continuity. In Judd's report in 1925, he stated that the method of hepaticoduodenostomy first developed by W. J. Mayo in 1905 had proved the most practical, the most widely applicable, and the most successful procedure for establishing the natural course of the flow of bile (Figs. 1, 2, 3 and 4).

I have used this type of procedure both in hepaticoduodenostomy and choledochoduodenostomy in 6 cases, in 5 of which excellent results followed, without further evidence of biliary obstruction. In the sixth case, Case 3 in the tabulation, severe intrahepatic cholangitis appeared 2 months after complete relief of biliary obstruction by choledochoduodenostomy. Subsequently, the liver, which had been cirrhotic at the time of operation, increased in size; the spleen became palpable and ascites occurred. By the use of one of the mercurial diuretics the ascites was controlled. When last heard from the patient had been free of fever and jaundice for several months (Fig. 2).

In connection with the prolonged good results following choledochoduodenostomy or hepaticoduodenostomy it should be noted that in one of these cases (Case 1)[1] a duodenal fistula developed immediately after operation, the toxæmia of which was controlled by the method described by Walters and Bollman. The patient has been perfectly well for more than 2 years, without the slightest evidence of obstruction or disease of the biliary tract (Fig. 3). This case has been reported in detail previously.

In another case (Case 2) choledochoduodenostomy was performed in August, 1927. Accumulation of bile around the liver depressed the organ and produced a chain of events characterized by extremely rapid pulse, increase in respirations, and semiconsciousness; this rapidly disappeared when the patient's wound was reopened in her room with the evacuation of the bile and the return of the liver and circulation to normal. A normal convalescence followed. The patient was allowed to return home 4 weeks after the operation; the wound was healed and her general health was excellent. She has been free of all jaundice, pain, or fever since the operation and feels perfectly well (Fig. 4).

PLASTIC OPERATIONS ON THE COMMON BILE DUCT

Following the report of McArthur's successful cases of plastic operations on the common bile duct in 1925, in which a catheter was used to serve as a scaffolding for healing and a means for transmitting bile, three plastic operations on the common duct were attempted in cases which were not well suited to this procedure. Instead of excising the stricture and making an end-to-end anastomosis, which the nature of the obstruction did not permit, the stricture was split longitudinally, allowing sufficient lumen of the duct, and then a closure was made transversely. In these 3 cases, the results of operation have been only fair. They have each presented at infrequent intervals evidence of what would seem to be incomplete biliary obstruction with occasional symptoms of transient jaundice or chills and fever with pain (Fig. 5).

[1] Case reported in detail in Surg. Clin. N. Amer., 1927, vii, 1576-1577.

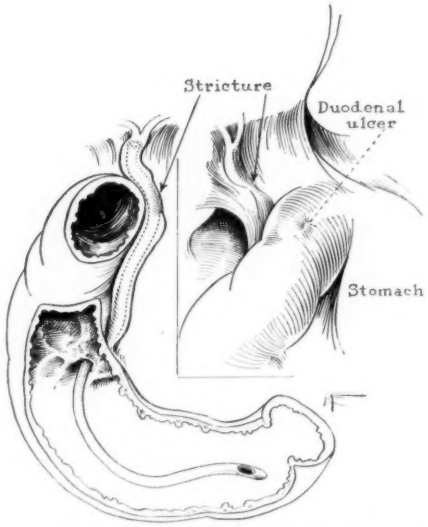

Fig. 5. Plastic operation for localized stricture of the common bile duct and excision of duodenal ulcer. Case 13.

In fairness to a discussion of plastic procedures on the duct itself, it must be said that an attempt was not made to secure union between the normal portions of the duct beyond the stricture since the stricture was not excised but rather to increase the lumen of the duct to normal or more than normal. However, since performing these three plastic operations on the duct, I have resorted entirely to the operation of hepaticoduodenostomy or choledochoduodenostomy, when a sufficient amount of duct proximal to the stricture existed for anastomosis to the duodenum, and the results have been excellent. On one occasion the stricture was confined to the upper portion of the common bile duct and involved the hepatic duct to such an extent that sufficient hepatic duct was not obtainable to anastomose to the duodenum. With plenty of normal common bile duct distal to the stricture, a plastic procedure was carried out February 7, 1928, after the method of the Heineke-Mikulicz pyloroplasty, a method which has been described as applicable to the common bile duct by both Moynihan and W. J. Mayo. An accurate anastomosis was made between the walls of the duct, rendering the size of

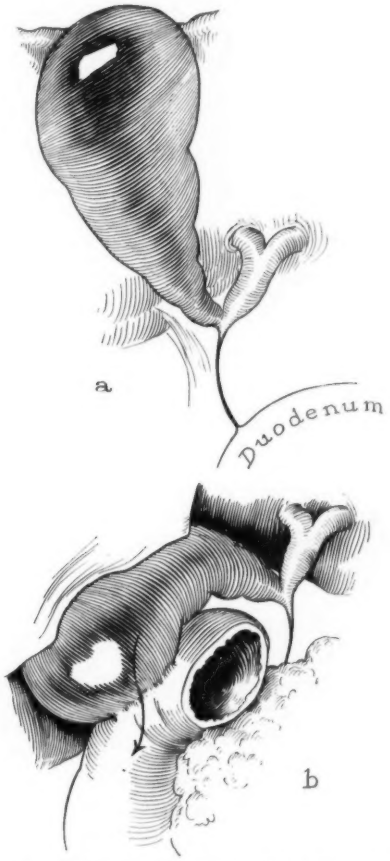

Fig. 6. Cholecystduodenostomy for stricture of the lower end of the common duct. Case 15.

the lumen even larger than normal. The patient's convalescence was complicated by the development of a subphrenic abscess, which was effectively drained (Harrington). Since operation, there has not been any evidence of biliary obstruction. In the discussion of this case it should be said that the method of plastic operation on the duct was employed as a method of necessity and not one of choice. It seemed to afford the only way of restoring continuity in the extrahepatic biliary passages.

The simplest methods of restoration of biliary intestinal continuity are those in which the gall bladder remains, and the stricture is

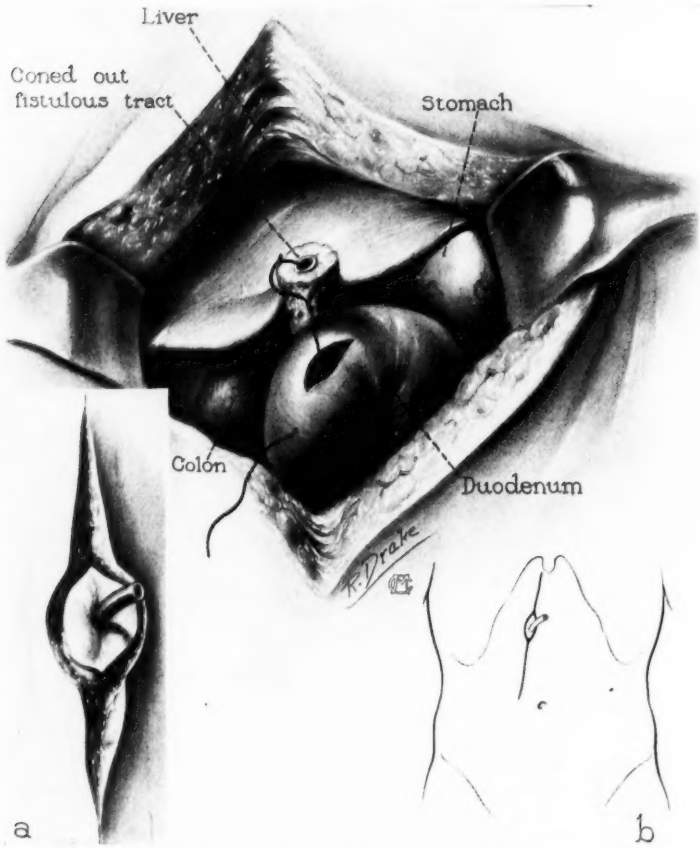

Fig. 7. There was complete stricture of the common and hepatic ducts. The illustration is of the coned-out external biliary fistulous tract ready to be transplanted into the duodenum. Case 8.

distal to the point of entrance of the cystic duct into the common bile duct. In these cases cholecystenterostomy is an easy solution of the problem. A woman aged 43 years who had been bedridden for almost a year subsequent to drainage of the gall bladder performed elsewhere has had a good result from cholecystoduodenostomy which I performed in January, 1926 (Case 15, Fig. 6). She has been working and feeling well since her operation except for transient periods of mild jaundice with fever lasting a day. These periods appear at intervals of several months and probably indicate the existence of residual cholangitis, which flares up at intervals. During the last 9 months symptoms referable to the biliary tract have been absent.

TRANSPLANTATION OF AN ESTABLISHED EXTERNAL BILIARY FISTULA

Cases of stricture of the common and hepatic ducts in which an insufficient amount of normal, common, or hepatic duct remains below the level of the liver to permit anastomosis to the duodenum, have constituted a surgical problem difficult to solve. The recent report, however, of successful transplantation of an established external biliary fistulous tract into the stomach or duodenum by Lahey, Masson, St. John, and Lilienthal has

served as an impetus to the use of this method in cases in which complete stricture of the extrahepatic biliary ducts exists. It should not be forgotten that the first successful transplantation of such a fistula was done by Williams at the Massachusetts General Hospital in Boston in 1914. The patient is still living and well. The ease with which such a coned-out fistulous tract can be transplanted into the duodenum is surprising. The only precaution necessary is that the external fistula shall have been present long enough so that it can be coned out as a well established tract and that it is left attached to the liver.

In one case in which I operated, establishing the external biliary fistulous tract in December, 1927, and transplanting the coned-out fistulous tract into the duodenum in March, 1928, the patient has been free from pain, jaundice, and fever; has gained in weight and feels well. The wound is solidly healed; stools are normal in color (Figs. 7 and 8, Case 8).

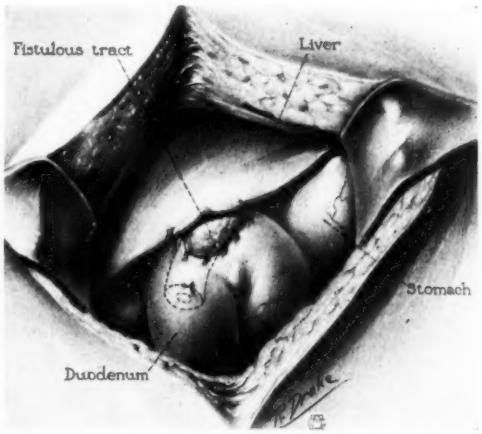

Fig. 8. Later stage in same operation as that shown in figure 7. Transplantation completed. Case 8.

SUMMARY

It has been shown by various observers that inflammation of the intrahepatic and extrahepatic biliary passages is associated with strictures of the common bile duct, and in many instances may be the predisposing factor to the development of the stricture. This factor, too, may account for the frequency with which incomplete intermittent obstruction occurs subsequent to plastic operations for the relief of strictures of the common or hepatic bile ducts in some cases.

A report of 17 cases of stricture of the common bile duct in which operation was performed during the last 4 years is presented with a description of the technique used as well as the progress in the months and years subsequent to operation. The operation of choledochoduodenostomy or hepaticoduodenostomy with an end-to-side or a side-to-side anastomosis, with an accurate union of mucous membrane of the duct to that of the duodenum, has proved to be the most satisfactory operation of the group. With this method excellent results have been obtained over a period of many months and in one case of more than 2 years.

The successful treatment of strictures of the common bile duct and the hepatic duct is dependent on the fact that sufficient duct remains proximal to the stricture to permit accurate anastomosis to an opening in the duodenum, as well as that a minimal amount of infection exists in the walls of the duct and the intrahepatic biliary passages.

In one case in which there was a very large anastomatic opening between the duct and the duodenum, severe cholangitis developed 2 or 3 months following the operation in the absence of extrahepatic biliary obstruction. It was accompanied by progressive enlargement of the liver and spleen and the formation of ascites. With the subsidence of the intrahepatic infection, jaundice and fever disappeared but the enlargement of the liver and spleen still persisted. The ascites, however, disappeared after the administration of a mercurial diuretic.

A case is reported in which the establishment of an external biliary fistula for complete stricture of the common and hepatic ducts, and the transplantation of the coned-out fistulous tract into the duodenum, was followed by a good recovery with relief of symptoms. The fistula was transplanted March 13, 1928, and the patient has been free of symptoms since. Six other successful cases of this type are reported in the literature.

SUMMARY OF RESULTS OF OPERATION

Hepaticoduodenostomy and Choledochoduodenostomy

Case	Age and sex	Date of operation	Serum bilirubin	Symptoms, duration	Condition of gall bladder, treatment	Lesion and operative procedures	Results of operation
1	63 M	5–15–26	17.4	Jaundice for 8 days	Cholecystostomy 1922	Stricture lower end of common duct; intrahepatic biliary calculi. Choledochoduodenostomy	December 1927 excellent; re-examined, no colics; no jaundice. 8–27–28 (letter) general condition remains excellent; stools normal, no jaundice.
2	44 F	8–2–27	6.3	Jaundice for 2 years	Cholecystostomy 1913 (elsewhere) Cholecystectomy 1923 (elsewhere)	Stricture lower third of common duct. Choledochoduodenostomy	1–21–28 excellent; gained 30 pounds; no jaundice. 8–25–28 general condition remains excellent.
3	25 F	9–30–26	13.6	Jaundice and colic for 3 months	Cholecystectomy 1926 (elsewhere)	Stricture lower portion of common duct. Choledochoduodenostomy	Jaundice, chills, fever, and enlargement of liver 6 months, postoperative cholangitis. 10–3–27 no jaundice; no ascites; large liver.
4	63 F	3–29–28	14.0	Jaundice and pain for 3 months	Cholecystectomy 1926 (elsewhere)	Stricture in common and hepatic ducts. Hepaticoduodenostomy	4–7–28 general condition excellent. 9–24–28 general condition excellent, no evidence of biliary obstruction.
5	50 M	8–2–28	3.3	Jaundice for 2½ months	Cholecystectomy 1927 (elsewhere)	Stricture in common and hepatic ducts. Hepaticoduodenostomy	8–22–28 excellent, dismissed.
6	64 F	6–30–25	4.7	Biliary fistula; no colics, slight jaundice	Cholecystectomy 1923 (elsewhere)	Stricture in common and hepatic ducts. Hepaticoduodenostomy	7–28–25 left hospital in good condition; died at home 20 months later.
7	36 F	9–4–28	14.0	Painless jaundice for 5 months	Cholecystectomy 1927 (elsewhere) Choledochogastrostomy 1927 (elsewhere)	Stricture in common and hepatic ducts. Hepaticoduodenostomy	Still in hospital.

Transplantation of External Biliary Fistula into Duodenum for Complete Stricture of Common and Hepatic Ducts

8	41 F	12–8–27 3–13–28	0.3	Pruritis and jaundice for 1 month	Cholecystectomy 1927 (elsewhere)	Common and hepatic ducts strictured to liver. External biliary fistula created. Transplantation of external biliary fistula into duodenum	4–11–28 excellent; wound healed, stools normal, no jaundice. 8–27–28 upper abdominal pain in July, no jaundice, chills, or fever; stools normal; gained 17 pounds.

Plastic Operation on the Common and Hepatic Ducts

9	50 F	4–20–26	1.7	Jaundice for 3½ years; dull epigastric pain	Cholecystectomy 1922 (elsewhere)	Stricture in common duct; reconstruction of duct	November, 1927, last attack of chills and jaundice. 3–6–28 gained 8 pounds. 8–29–28 no pain, fever, chills or jaundice.
10	49 F	5–10–27	1.0	Colic for 4 years	Cholecystostomy	Incomplete stricture at juncture of common and hepatic ducts; sandy material in hepatic duct; dilatation of stricture. Choledochostomy	10–6–27 returned on account of hernia; no symptoms from common duct. 8–29–28 biliary obstruction and pain 3 days' duration. Backache and vomiting and slight jaundice; stools normal; no fever.
11	41 F	8–6–25		Colic for 5 months followed by jaundice	Contracted and formed small fibrous mass 1.5 cm. indiameter	Postoperative incomplete stricture of common duct at cystic duct. Plastic on common duct	12–16–26 return of jaundice with slight pain. 9–20–28 pain and jaundice absent since 1926, occasional short period of chills and fever.
12	48 F	3–6–26	12.8	Splenomegaly, biliary cirrhosis	Cholecystectomy 1925 (elsewhere)	Stricture of hepatic duct and stones in common duct. Reconstruction of duct	3–13–26 died in hospital; necropsy showed cirrhosis of liver, splenomegaly, intra-abdominal hæmorrhage, suppurative cholangitis.
13	39 F	7–30–25	9.0	Colic for 2 years	Cholecystectomy (elsewhere)	Stricture of common duct, 2 cm. long. Plastic reconstruction of common duct over catheter. Excision duodenal ulcer	April, 1928 recurrence of biliary obstruction and jaundice. 8–29–28 intermittent jaundice but no pain, chills, or fever.
14	32 M	2–7–28	3.8	Intermittent colic and jaundice for 2 years	Cholecystectomy 1924 (elsewhere)	Incomplete stricture of middle and upper thirds of common and hepatic ducts. Hepaticocholedochostomy	5–14–28 free of jaundice; no fever, general condition good.

Cholecystduodenostomy

15	43 F	1–14–26	3.8	Colic and jaundice for 2½ years; in bed 1 year	Cholecystostomy 1923 (elsewhere); distended and contained thick dark bile	Stricture of common duct. Cholecystduodenostomy	3–14–28 good result. Occasional slight jaundice. 8–27–28 no further jaundice, stools normal; no pain or fever. Slight hæmatemesis on 7–22–28 of unknown origin.

SUMMARY OF RESULTS OF OPERATION—Continued

CHOLEDOCHOGASTROSTOMY

Case	Age and sex	Date of operation	Serum bilirubin	Symptoms, duration	Condition of gall bladder, treatment	Lesions and operative procedures	Results of operation
16	49 F	7–22–27	1.5	Jaundice, itching, and gas pains since 1922	Cholecystectomy 1922 (elsewhere)	Stricture of common and hepatic ducts. Stone in hepatic duct. Choledochogastrostomy; T-tube in common duct	10–12–27 good result, tube passed. 12–8–27 return of chills and jaundice. 8–28–28 jaundice, chills, and fever; no pain.
17	39 F	7–25–28	10.0	Jaundice and itching 2 months	Operation for perforated duodenal ulcer April, 1927 (elsewhere). November, 1927 Cholecystectomy (elsewhere)	Almost complete stricture of common and hepatic ducts 1 cm. in width remaining	Died in hospital 12 hours postoperative; intra-abdominal hæmorrhage.

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MALIGNANT TUMORS OF THE FEMALE BREAST

A Clinical and Pathological Study of Two Hundred and Thirty-Four Cases From the Clinic of the Free Hospital for Women

GEORGE VAN S. SMITH, M.D., and MARSHALL K. BARTLETT, M.D., Boston

THE material for this report was obtained by a clinical pathological study of 234 cases of malignant breast tumors, 197 of which were treated at the Free Hospital for Women, Brookline, Massachusetts, between 1875 and 1928, and 37 of which were treated in the private practice of William P. Graves and Frank A. Pemberton between 1909 and 1928. Seven cases, 2.9 per cent, were diagnosed sarcomata; the rest were carcinomata. The diagnosis was made or confirmed by microscopic examination in all but 5 cases and these were clinically unmistakable.

AGES

The patients were classified into five-year age groups. Five were under 30 years of age, the youngest being 22; and 8 were over 75, the oldest being 83. Between these two extremes the total number of cases in each age group reached a maximum in the 45-to-50-year-old group. Nearly three-fifths of the patients of this series were between the ages of 45 and 65.

FAMILY HISTORY

Of the 197 free clinic patients, 27 gave a family history of tuberculosis and 33 (16.7 per cent) gave a family history of malignant disease.

PAST HISTORY

Fifteen patients gave a past history of breast trauma. In most of these it seemed evident that the trauma had served to draw the patient's attention to the lump. Four patients had had abscesses of the same breast and later carcinomata. In these patients the breast had been lanced respectively 7, 10, 10, and 15 years previously. In one the cancer had clearly originated in the abscess scar. In three instances a tumor had been excised from the same breast 25, 12, and 9 years respectively before cancer was diagnosed. The last of these three patients had also had a tumor excised from the other breast 15 years previously. One patient had had a tumor excised from the other breast 7 years before admission; one had had a radical amputation of the other breast 18 years before admission, presumably for cancer.

MARITAL

Seventy-nine patients, 33.7 per cent, had never been pregnant. Fifty-three of these were unmarried. Of the 176 married patients 7 had had only abortions or miscarriages and 56, 23.9 per cent, had had only one child. Thus 36.7 per cent of this series had never nursed. There is no data as to how many of the patients with children had never nursed. The average number of children per married patient was 2.3. These findings agree with those of Lane-Claypon who compared 500 women with cancer of the breast with a control series of normal women. To quote from her report: "The incidence of cancer of the breast is greater among single women and the less fertile married women, i.e., those in whom the gland does not attain full function."

SYMPTOMS AND DURATION

It is common knowledge that the first symptom in this disease is most often the finding of a lump in the breast. This was so in 230 of this series. Four complained of bleeding and 5 of discharge from the nipple. The duration of symptoms varied from 2 weeks to 32 years. Seven patients had had symptoms 4 to 5 years, 6 had had symptoms 5 to 6 years, 4 for 7 to 10 years, and 3 from 20 to 32 years. It seems reasonable to assume that a benign tumor preceded the malignant neoplasm in those patients who had noticed a lump for over 7 years.

TREATMENT

It is generally conceded that the radical operation of removing breast, both pectoral muscles, axillary contents, and deep fascia *en*

masse as early as possible, in properly selected cases, is the procedure of choice. "Properly selected" means that there must be no palpable supraclavicular glands, no adherent axillary glands, no internal or bone metastases and that the tumor must be at least movable on the chest wall. When the operative risk is great because of some other disease or senility, or when there is ulceration and infection of the breast in an advanced case, simple amputation without removal of the pectoral muscles or axillary contents may be preferred. In the present series 225 patients were treated by operation, simple amputation of the breast being performed in 26.2 per cent of cases, and radical amputation in 73.8 per cent. There were 3 operative deaths due respectively to diabetic coma, cerebral hæmorrhage, and pulmonary embolus (1.3 per cent).

GROSS STATISTICS

Nine patients were not treated because of the advanced stage of the disease when seen on admission. One is untraceable, 7 died 5 months or less after being seen, and one died one year and two months later. The course of the disease from the time symptoms were first noticed until death varied from one year and one month to 7 years and 3 months. In 5 cases the course of the disease was less than one year and 8 months; in 2 it was 5 years and 3 months, and 7 years and 3 months, respectively. The course of the disease in the 225 remaining cases is shown in Table I.

Untraceable.—20

TABLE I.—COURSE OF DISEASES IN TWO HUNDRED AND TWENTY-FIVE CASES

Postoperative period	Died	Living with recurrence	Well
One year or less	34	8	8
One to two years	30	5	3
Two to three years	22	5	2
Three to five years	19	4	17
Five to seven years	12	4	6
Seven to ten years	5	1	7
Ten to fifteen years	4	.	4
Fifteen to twenty years	..	.	5

Of the 166 patients traceable at the end of 3 years, 88 (53 per cent) were alive. Of the 130 traceable at the end of 5 years 48 (36.9 per cent) were alive. Of 101 traceable at the end of 7 years 26 (25.7 per cent) were alive.

RESULTS OF SIMPLE AND RADICAL AMPUTATION

After examining 20,000 cases reported in the literature of various countries, Lane-Claypon found that after incomplete operation 29 per cent of cases were alive after 3 years, while after radical or complete operation 43 per cent survived the three-year interval.

Greenough, in studying 135 cases at the Massachusetts General Hospital for the years 1918 to 1920, inclusive, reported 30 per cent of 5-year cures following radical operation.

Table II compares the results of radical and simple amputation in the present series:

TABLE II.—RESULTS OF RADICAL AND SIMPLE AMPUTATION

	Radical amputation	Per cent alive	Simple amputation	Per cent alive
Traceable 3 years after operation	128	52.3	38	55.2
Traceable 5 years after operation	100	37	30	36.7
Traceable 7 years after operation	84	27.4	17	17.6

Oddly enough, although Lane-Claypon reports that 29 per cent of cases treated by simple amputation were alive at the end of 3 years, of the 38 cases in the present series traceable 3 years after simple amputation 55.2 per cent were alive. This discrepancy can be accounted for only by the fact that a number of simple amputations were done on early cases and that some of the tumors were of a less malignant nature.

THERAPEUTIC X-RAY

Daland has reported figures showing that in cases treated pre-operatively with X-rays, defective wound healing occurred in 54 per cent, while in cases untreated with X-ray before operation defective healing occurred in only 18 per cent.

Regarding postoperative X-ray therapy, Greenough reported 21 cases of radical amputation without X-ray treatment who lived an average of 32 months after operation, and 29 cases of radical operation followed by X-ray who survived for an average of only 21 months.

In contrast to this the results in the present series were uniformly better after postoperative X-ray treatment, as indicated in Table III and later in this report:

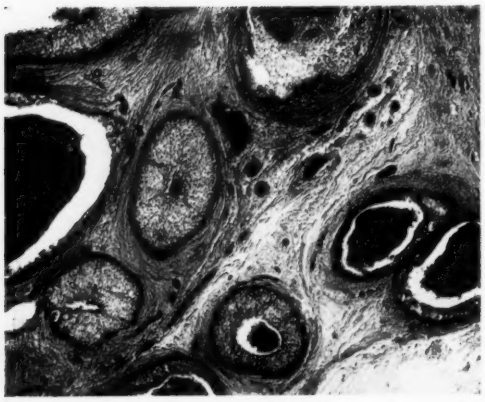

Fig. 1. Photomicrograph of a carcinoma of the breast of low malignancy, class I. The patient was alive and well 6 years and 5 months after a radical amputation, having had no X-ray treatment. There was no glandular metastasis at the time of operation. The alveoli show central necrosis and are well surrounded by fibrous tissue. There is no round cell infiltration.

TABLE III.—RESULTS OF POSTOPERATIVE X-RAY TREATMENT

	Post-operative X-ray	Per cent alive	No Post-operative X-ray	Per cent alive
Traceable 3 years after operation	52	69.2	98	40.7
Traceable 5 years after operation	37	51.4	83	27.7
Traceable 7 years after operation	20	30	73	20.5

(This table included both simple and radical amputation cases.)

Table IV is composed only of cases treated by radical amputation.

TABLE IV.—RESULTS AFTER RADICAL AMPUTATION

	Post operative X-ray	Per cent alive	No Post-operative X-ray	Per cent alive
Traceable 3 years after operation	37	64.9	80	43.8
Traceable 5 years after operation	18	72.2	69	30.5
Traceable 7 years after operation	10	60	63	22.2

RADIUM

It is conceded by most authorities that radium is of little or no use as a primary treatment for this type of cancer. For inoperable cases and recurrences X-ray is considered to be the most effective treatment, although radium has definite value in destroying superficial recurrent nodules. In 3 of 4 cases of this series, in which it was used for this purpose, radium brought about the complete disappearance of recurrent nodules.

MICROSCOPIC DEGREE OF MALIGNANCY

In recent years, the literature has contained reports of series of breast cancer in which each case was classified as to degree of malignancy on the basis of the microscopic picture in order to determine whether or not a prognosis could be made in this manner. Greenough reported a series of 90 cases, classified into four grades of malignancy by microscopic examination. He found that after 3 years 66 per cent of the class I or low malignancy group were living. After the same interval 47 per cent of the class II group and 23 per cent of the class III group were alive, while none of the class IV, or highest malignancy group, had survived.

To gauge the degree of malignancy by microscopic examination requires considerable experience in the study of many sections. In general the criteria determining high as against low malignancy are: lack of cell differentiation and uniformity; variation in size, shape, and staining reaction; relative frequency of mitoses; lack of round cell infiltration and walling-off connective tissue proliferation; invasion of surrounding tissues and absence of regular cellular conformation and groupings, e.g., tubules (see Figs. 1, 2, and 4). It should be mentioned that at best the determination of the degree of malignancy is most difficult, for it is rare to find a breast tumor the cells of which are homogeneous throughout. Usually pictures of varying malignancy can easily be found in the same tumor, and frequently a metastasis has an entirely different appearance from that of the primary growth (see Figs. 3 and 3A). Clinically the same tumor seems to vary in its malignancy, for hopeless cases may live a surprisingly long time, while cases with a good prognosis may die from rapid, unexpected recurrence. Furthermore, late recurrences may not have the slightest resemblance in degree of malignancy to the primary tumor. The gauging of the degree of malignancy, then, is an estimate as to prognosis based on experience in examining many breast cancers microscopically.

Two hundred and five of the cases herein reviewed were graded according to their malignancy as determined by microscopic study, class I being low and class III high malignancy. At the time of grading the tumors

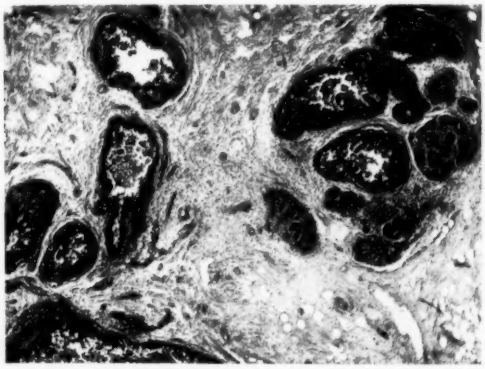

Fig. 2. Class I carcinoma of the breast. The patient is well 10 months after a radical operation at which were found axillary gland metastases. This picture was taken to illustrate carcinomatous metaplasia in an area of chronic mastitis. There is considerable defensive fibroblastic proliferation and round cell infiltration.

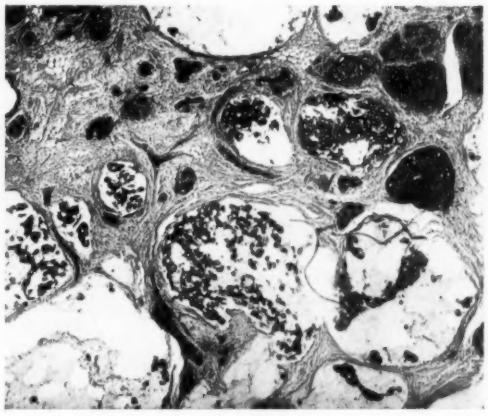

Fig. 3. Class I carcinoma of the breast. Simple amputation of left breast in 1909; simple amputation of right breast in 1910. In 1912 recurrent nodules were excised from left chest wall and the left axilla was dissected. Treatment by X-ray then followed. The patient was alive and well in 1927, 18 years after the first operation. The picture shows chronic mastitis and a carcinoma of low malignancy. The carcinoma looked like the comedo type in some sections; here it is more of a colloid type.

pathologically the clinical records of the patients were not consulted. The late Dr. J. H. Wright, Dr. H. F. Hartwell, and Dr. Albert E. Steele very kindly examined nearly one-fourth of the sections; Dr. R. B. Greenough very kindly examined a number of sections. One of the writers (Smith) examined nearly all of the sections a second time, after a lapse of over 2 years, and agreed with the previous gradings in nearly every instance. (Table V).

TABLE V.

	Class I.	Per cent alive	Class II.	Per cent alive	Class III.	Per cent alive
No. traceable at 3 years...	9	88.8	77	59.8	65	40
No. traceable at 5 years...	8	87.5	64	42.2	44	13.6
No. traceable at 7 years...	6	83.3	49	28.6	35	8.6

AXILLARY GLANDS

A study of prognosis based on the presence or absence of axillary gland metastasis was made in classes II and III. Of the three class I cases with gland metastasis one died 9 years 10 months after operation, and 2 were well respectively 8 months and 17 years and 6 months, after operation. The survival figures in relation to gland involvement and grade of malignancy are shown in Table VI, which is made up only of cases treated by radical amputation—some having had post-operative X-ray.

TABLE VI.—GLAND INVOLVEMENT AND GRADE OF MALIGNANCY

	Group II.				Group III.			
	Glands involved	Per cent alive	Glands not involved	Per cent alive	Glands involved	Per cent alive	Glands not involved	Per cent alive
Traceable 3 yrs. after operation	29	48.4	26	84.5	42	26.3	9	89
Traceable 5 yrs. after operation	27	25.9	21	66.7	29	6.9	5	60
Traceable 7 yrs. after operation	22	13.6	17	52.9	26	4.7	4	50

GRADE OF MALIGNANCY AND DURATION OF SYMPTOMS

Since the duration of symptoms before operation is at best an inaccurate figure, based as it is on individual variation, and since it is a factor over which the surgeon has little control, no attempt has been made in this report to correlate it with the grade of malignancy and results, although it is accepted to be a factor of great importance.

DEGREE OF MALIGNANCY AND X-RAY TREATMENT

The cases in classes II and III treated by radical amputation with and without the use

of the X-ray after operation were correlated with the degree of malignancy (Table VII).

TABLE VII.—DEGREE OF MALIGNANCY AND X-RAY TREATMENT

	CLASS II.				CLASS III.			
	X-ray	Per cent alive	No X-ray	Per cent alive	X-ray	Per cent alive	No X-ray	Per cent alive
Traceable at 3 years	22	63.6	34	61.8	11	63.6	37	16.2
Traceable at 5 years	16	62.6	41	29.3	5	20	30	10
Traceable at 7 years	12	41.6	30	30	2	50	29	6.9

Although these figures cover only a few cases, they indicate not only that postoperative X-ray treatment is of value in all cases, but that its value is even greater in the more malignant grades.

SARCOMA OF THE BREAST

There were 7 cases of sarcoma of a total of 234 cases, 2.9 per cent. Three of these had never been pregnant and two had had only one child. The patients had noticed a lump in the breast for periods varying from 7 months to 8 years. One patient died of pulmonary embolus after operation; two are untraceable; one was alive with an advanced recurrence 5 months after operation; one died 1½ months after operation; one died 5½ years after operation, having had twenty-seven operations in all for the primary growth and for recurrences; and one patient was well 11 years after operation. On pathological examination 5 of these tumors were found to be fibrosarcomata, and two, round cell sarcomata. In 6 instances unmistakable intracanalicular or pericanalicular adenofibromata were found closely associated with the sarcomata.

ASSOCIATED PATHOLOGY

Chronic mastitis was practically a constant associated finding in the same breast with the malignant tumor. "Chronic mastitis," however, is found so frequently in breast tissue and its microscopic appearance varies so widely that its significance cannot be estimated. In 5 cases tumors removed from the other breast, at the time of the operation for cancer, showed chronic mastitis. Five patients had papillary cystadenoma in the same breast with the cancer (1 was class I malignancy, 4 were class II) and one of these had a papillary cystadenoma in the other breast as well. In another case papillary cystadenoma was found in the other breast which had been removed at the same time as that containing the carcinoma. In studying many sections one gets the impression that chronic mastitis, papillary cystadenoma, and carcinoma are stages in the same process. Although, in the study of benign breast tumors, it was common to find chronic mastitis and a pericanalicular or intracanalicular adenofibroma in the same breast, not once in this series was a pericanalicular or intracanalicular adenofibroma found associated with a carcinoma.

Twelve patients had the other breast amputated for carcinoma from 1 month to 4 years after the primary operation. So far as could be determined these were metastatic carcinomata. One patient had carcinoma of the cervix at the time of diagnosis of breast carcinoma; one had carcinoma of the endometrium 6 years later.

SUMMARY AND CONCLUSIONS

1. This report covers a clinical pathological study of 234 malignant tumors of the female breast of which 2.9 per cent were sarcomata, the remaining being carcinomata.

2. Nearly 60 per cent of patients were between the ages of 45 and 65 years.

3. A family history of malignant disease was given by 16.7 per cent of patients.

4. Nine patients had had previous breast operations, only one of which was for carcinoma, so far as could be determined.

5. The average number of children per married patient was 2.3. It was found that 23.9 per cent of patients had had only one child and that at least 36.7 per cent of the entire series had never nursed.

6. Practically all of the patients complained of finding a lump in the breast.

7. The duration of symptoms was usually under 2 years. Seven patients had had symptoms for more than 7 years, which makes it probable that a benign tumor antedated the malignant disease.

8. In untreated cases the course of the disease varied from 1 year and 1 month to 7 years and 3 months.

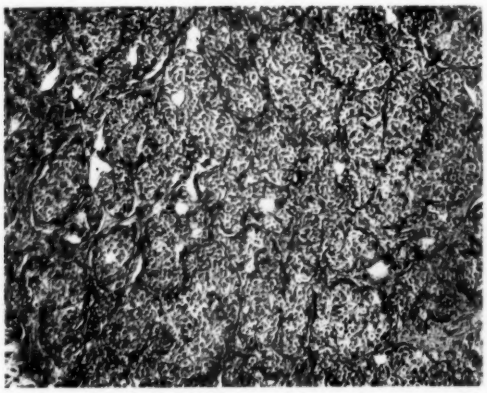

Fig. 3A. Section of left axillary gland—same patient as in Figure 3. This shows how different a metastasis may appear from the primary growth. Neither the cells nor their arrangement in this section resemble the cells or conformation of the primary growth. This also appears more malignant than the primary growth.

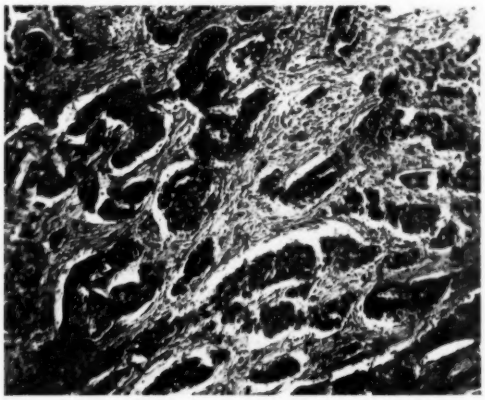

Fig. 4. Section showing class III, high malignancy type of carcinoma of the breast. There is very little histologic defense against the invading masses and alveoli. The cells vary in size, shape, and staining reaction, and mitoses can frequently be found under high power objective. The patient had had symptoms for 5 months; she died 5 months after a radical amputation.

In many cases it is very difficult to decide whether a case should be graded class III or class II, but after studying many sections, one learns to detect differences. Photomicrographs do not bring out these differences with any satisfactory degree of clearness.

9. The operative mortality was 1.3 per cent.

10. In this series 53 per cent of all operated upon, traceable cases were alive at the end of 3 years, 36.9 per cent were alive at the end of 5 years, and 25.7 per cent were alive at the end of 7 years. The percentage of absolute cures cannot be determined because some patients are untraceable, and because recurrences may occur years after an apparent cure. Of the 13 patients known to have lived longer than 10 years after operation 2 died of recurrent carcinoma between 11 and 12 years after operation.

11. Contrary to expectations, the 3 and 5 year results in those cases treated by simple amputation were practically the same as in cases treated by radical amputation, although the 7 year results were not as good. Since this finding is exceptional and probably a coincidence, it should not be construed to favor simple in preference to radical amputation.

12. The degree of malignancy may be determined in any given case on the basis of the microscopic examination, and an approximate prognosis may be made, due consideration being given to the duration of symptoms, to the stage of the disease when seen, to the presence or absence of axillary metastasis, and to the ability of the operator. In this series there survived the 7 year interval 83.3 per cent of class I cases, 28.6 per cent of class II, and 8.6 per cent of class III cases.

13. In classes II and III the finding at operation of axillary gland metastasis affected the prognosis markedly.

14. After postoperative X-ray treatment, the results were uniformly better both as to gross figures, to type of operation, and to degree of malignancy.

15. In 6 of the 7 cases of breast sarcoma, an intracanalicular or pericanalicular adenofibroma was found closely associated with the malignant tumor.

16. Although chronic mastitis is almost a constant finding in carcinoma of the breast, it cannot be shown to have any etiological relationship. On the other hand, there is frequent evidence that the papillary cystadenomata are often precursors of a malignant condition.

NOTE.—In this paper the study of the grades of malignancy were undertaken at the instigation of Dr. R. B. Greenough, and to him, the late Dr. J. H. Wright and Dr. H. F. Hartwell for their interest and assistance the writers express their gratitude.

To Dr. W. P. Graves, who operated on 59.9 per cent of the cases in this series, and to Dr. F. A. Pemberton who operated on 27.1 per cent, the writers are grateful both for permission to report these cases and for much interest and co-operation.

Most of the X-ray treatments received by patients in this series were given by Dr. L. B. Morrison, to whom the authors express their thanks for co-operation and assistance.

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THE MANAGEMENT OF URETERAL INJURIES WITH A DISCUSSION OF THE SURGICAL INDICATIONS IN PATIENTS WHO REQUIRE URETERAL TRANSPLANTATION[1]

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From the Gynecological Department of St. Luke's Hospital.

Frank Billings has an axiom to the effect that the best way to secure a satisfied patient is to find out what he wishes and then treat him in accordance with his desires. I know very well what everyone in this audience would wish from me were he empowered to speak—it would be a carefully concentrated paper, free from exhaustive explanations and discussion.

Introductory to the substance of my offering, I wish to call to your attention that three men here tonight have done a major portion of the world's greatest work in ureteral surgery. Franklin H. Martin, unexcelled benefactor in the advancement of medical organization, through his pioneer work in animal experimentation evolved a method of ureteral implantation into the bowel which, in its chief features, embodies the principles of Coffey's technique. The experimental and clinical work of Dr. Coffey and the achievements of Charles Mayo in human subjects require no emphasis at this time. The outstanding work of E. Starr Judd, William E. Lower, and Arthur L. Chute also deserves special recognition. The experimental work of C. M. McKenna, which has just been reported to you here tonight, is of unusual interest and importance.

MANAGEMENT OF THE INJURED URETER

What shall be done with a severed ureter, the injury being recognized at the time of the operation?

1. If a ureter is divided during the course of a hazardous or markedly prolonged operation, and it is chiefly in such cases that this calamity occurs, I am unqualifiedly in favor of ureteral ligation, without attempt at transplantation or repair. (It is assumed that palpation reveals an apparently healthy kidney and ureter on the opposite side.) Experience has shown that patients subjected to ureteral ligation rarely require subsequent surgical intervention and life expectancy is not materially shortened.

2. If the operation has *not* been notably hazardous, also in selected hazardous cases in which there is evidence of notable kidney-ureter pathology on the uninjured side, restitution of function of the injured ureter appears indicated.

Transplantation of the ureter into the urinary bladder is the accepted procedure of choice in those unusual cases in which the divided ureter is of sufficient length to permit implantation without tension. As a rule this is not feasible.

An apparently ideal method of management of a severed ureter is restitution by end-to-end anastomosis (by interrupted fine catgut sutures which do not enter the lumen) over a *snugly-fitting* fine rubber tube (e.g., a small urethral catheter), the upper end of which emerges from a slit in the ureter above the anastomosis and projects through a stab wound in the flank. In women the tube need not emerge from a ureteral slit; instead, the

[1] Read before the Clinical Congress of the American College of Surgeons, Boston, October 9, 1928.

Fig. 1. Photograph of the kidneys and ureters of dog killed 9 months after operation. *a*, Site of suture of the right ureter. (Courtesy of Drs. Bump and Crowe.)

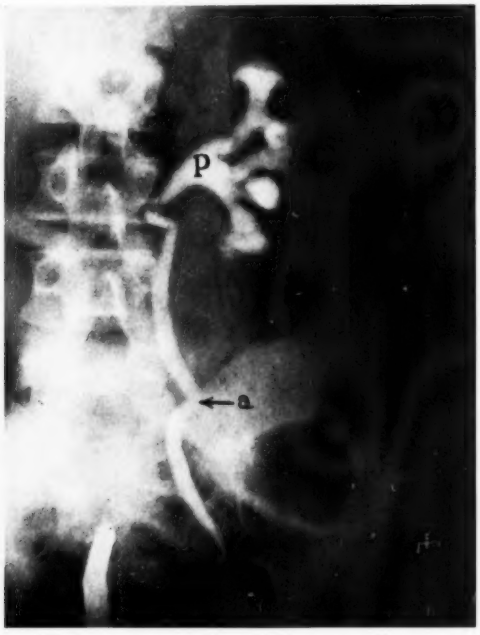

Fig. 2. Pyeloureterogram of patient 5 months after anastomosis of severed ureter. *P*, Normal kidney pelvis; *a*, site of ureteral anastomosis.

lower end may extend downward into the bladder, from which it is subsequently removed through the urethra.

Temporary deviation of the urinary stream above the anastomosis is essential to success. This is accomplished by means of another fine rubber tube, or small urethral catheter, which is inserted through the ureteral slit and extends upward in the direction of the kidney. This tube also projects through the stab wound in the flank and drains the entire urinary stream until removal of both tubes, eight or ten days later.

The feasibility of this procedure has been demonstrated experimentally, beyond all question, by Warner S. Bump, of the Gynecological Department of Northwestern University Medical School, in conjunction with Stanley M. Crowe. Their work was undertaken at the suggestion of Dr. L. L. McArthur, who performed the first operation upon a human subject. Of a total of six dogs which survived operation and in which the drainage tubes remained in position, every one recovered without urinary tract infection and with perfect restitution of ureteral continuity, without necropsy evidence of ureteral stricture (Fig. 1). Of the control animals, in which urine was permitted to flow through the normal channel during convalescence, all developed stricture of the ureter at the point of anastomosis.

This technique has been employed in one of the two cases of cut ureter which have occurred on our service during the last year. The patient was operated upon in November, 1927. Pyeloureterography five months after operation, and again after ten months, revealed restoration of ureteral continuity and a normal kidney pelvis (Figs. 2 and 3). Dye tests, urine examinations, and urine cultures indicate normal kidney function. I believe that this is the only recorded case in which end-to-end anastomosis of the ureter has been followed by clinical proof of normal kidney-ureter function.

Fig. 3. Pyeloureterogram of patient 10 months after anastomosis. Normal kidney function. *a*, Anastomosis.

What shall be done with an injured ureter, the injury being first recognized during postoperative convalescense?

It is impossible to estimate the percentage of deaths ascribable to intra-abdominal extravasation of urine resultant from ureteral injury; it is considerable. We all know, however, that a majority of these patients live, that they drain, and that they are continuously uncomfortable.

I wish to emphasize that these patients with leakage of urine tend to progress to spontaneous cure due to the gradual development of ureteral stricture. In experimental animals, the flow of urine is practically always insufficient to prevent spontaneous closure of the injured ureter. In human subjects the results are not so ideal but watchful waiting usually suffices. Restriction of liquid intake and careful manipulation at the supposed site of injury are possible aids in promoting closure.

In the event that surgical intervention eventually becomes necessary, the indications for operation vary greatly according to the individual case. In an otherwise healthy patient, not notably obese and probably free from extensive adhesions, abdominal section may be given preference over nephrectomy. My procedure of choice in such a case would be ureteral anastomosis. That being impossible, in the presence of a healthy second kidney and ureter, I would ligate the ureter. It is my belief that future experience will condemn the intestinal transplantation of an injured ureter in patients who have a normally functionating, apparently healthy kidney and ureter on the opposite side.

PROBLEMS IN URETERAL TRANSPLANTATION

I most heartily approve of ureteral transplantation and am a warm advocate of Coffey's original technique in the performance of this operation.

A limited personal experience indicates that the cases which require transplantation are relatively rare. This statement needs special emphasis because the operation is a difficult and dangerous one in the hands of those who perform it infrequently. The chief indications for ureteral transplantation are irreparable injuries of the bladder, total cystectomy, cystitis dolorosa, and intolerable inflammation of the bladder incident to the presence of residual urine. Unilateral ureteral injury is excluded from this group.

Simultaneous transplantation of both ureters has not been attempted on our service at St. Luke's Hospital because we have felt that it is too hazardous. Added experience, on the part of others, may change our views.

In closing, I wish to direct your thoughts to certain features of technique and certain queries which arise:

Preliminary ureteral catheterization, when feasible, expedites finding the ureters.

The Trendelenburg posture, with the bowel well walled off by a rubber pack, simplifies the operation.

Obese and barrel-shaped patients are particularly unfavorable subjects.

A ureter which is under tension when it is transplanted offers a serious menace; leakage of urine means possible death of the patient.

Despite gentlest manipulation and most careful technique, transplantation of a ureter is often followed by extensive adhesions. In the event that operation for transplantation of the second ureter reveals bad adhesions, also in patients who are poor surgical risks, likewise in those who, for various reasons, are difficult to operate upon, it may be desirable to be satisfied with a single transplanted ureter. In fact, serious consideration should always be given to the possible desirability of ligating the second ureter provided palpation from within the abdomen confirms other indications of satisfactory function on the already transplanted side. Particularly in case of anticipated total cystectomy for bladder cancer, it would appear most logical to make preliminary transplantation of one ureter, with subsequent ligation of the second ureter at the time of removal of the bladder.

A final word of warning: Ureteral transplantation into the sigmoid or rectum offers an excellent cure for constipation. A painless liquid evacuation occurs every several hours. Occasionally, however, there is persistently delayed evacuation or partial retention of fæces and urine with associated resorption of toxic urinary products from the bowel. Repeated colonic flushings prevent the development of chronic uræmia in these cases.

CONCLUSION

Uretero-ureteral anastomosis is an excellent procedure in selected cases of ureteral injury. Ureteral transplantation into the intestine, according to the technique of Coffey, is of great value in patients in whom the bladder is no longer serviceable as a urinary reservoir. Although both of these operations are of inestimable value, it must be borne in mind that the cases in which they are indicated are relatively rare.

MALIGNANT BONE TUMORS[1]

PROFESSOR VITTORIO PUTTI, BOLOGNA, ITALY

THE American College of Surgeons has an established tradition whereby the work of its annual Congress shall be inaugurated by the pious rite of evoking the memory of a great surgeon and famous master, John B. Murphy. The Board of Regents has this year decreed that this signal honor should be entrusted to me, and it is with deep respect for this important mission that I have come to offer my humble contribution.

But at the actual moment in which I prepare to fulfill my undertaking, I feel that I must appeal to your courtesy and indulgence. I cannot offer a perfect eulogy of John B. Murphy, because I never had the privilege of meeting him. I cannot entertain you with a subject of general interest because I am the modest devotee of one specialty. For these reasons I hesitated long before I accepted the generous and cordial invitation of your Director General, but my diffidence was ultimately overcome by the desire not to let slip so favorable an opportunity of demonstrating both my profound admiration for this great American surgeon and also my gratitude as an Italian to the American College of Surgeons for honoring my country a second time by entrusting the task of delivering the Murphy Oration to a surgeon of my country.

I believe that it is not necessary to have known John B. Murphy in order to appreciate the great rôle which he played in the evolution of modern surgery. Certainly those, like myself, who were not privileged to meet him, cannot but have missed the illumination which radiated from his complex personality and the profitable example of his exceptional skill. But his real reputation, that for which he is admired as one of the most illustrious exponents of modern surgery, is founded on his writings. Every one of his contributions either opens up a new field or clears up some confused subject. He is perhaps the last of a generation of general surgeons who were able to cope with the whole field of medical practice, in all its complexities, and who have laid the foundations of modern specialization. Murphy was a pioneer in every branch of surgery, but, as Bastianelli has already remarked, in none did he find greater opportunity to demonstrate his genius than in the field of the surgery of the bones and joints. In this he was not only a pioneer but also a constructor, and I believe that, were he among us, it would not be unpleasant to him to find his memory honored by a discussion on one of the subjects which interested him most, and in connection with which the American College of Surgeons, the heir of his thoughts and aspirations, has created that admirable research organization, which goes by the name of the American Registry of Bone Sarcoma.

I hope that you will not accuse me of presumption if I venture to lay before you my views on a subject which has been studied in your midst by surgeons such as Bloodgood and Coley and by pathologists such as Codman and Ewing, and also if I dare to draw my conclusions from statistics which cannot be compared with those utilized by Kolodny. It is only because of my admiration for the work of American surgeons and pathologists that I wish to give to you the fruits of my own experience in the hope that they may prove of use in the solution of a problem to which your countrymen have devoted so much energy and knowledge.

DIAGNOSIS

It is my opinion that the problem of neoplasms of the skeleton, *clinically considered*, is first and foremost a problem of diagnosis. We must confess at once that, if we know nothing of the essence and causes of tumors in general, in the case of bone tumors we have not even a clear clinical picture.

The name of *osteosarcoma* covers an ill-defined, pathological condition, of which we know only a few really characteristic features; that of *giant-celled* tumor refers to a pathological growth, which is not yet known to belong for certain to the neoplastic diseases, which is considered benign, yet which often

[1] The John B. Murphy Oration in Surgery, delivered before the Clinical Congress of the American College of Surgeons, Boston, October 8, 1928.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Osteogenic sarcoma. Sun-ray arrangement of the spicules of the new formed bone.

Fig. 2. Endothelial myeloma (Ewing's sarcoma). Note the location in the shaft, the onion-like arrangement of the layers of the new formed bone and the diffuse involvement of the marrow.

Fig. 3. Endothelial myeloma. Although tumor affects three vertebræ, intervertebral discs remain intact.

cannot be distinguished from a malignant blastoma. Primary and secondary tumors assume the appearance of inflammatory processes, in the same way that infections and dystrophic diseases imitate the structure of a neoplasm.

So much for the diagnostic problem, considered by itself. But we all know that, in respect to malignant tumors, early diagnosis is still the only certain way of ensuring a radical cure. Therefore, we repeat, the problem is primarily one of diagnosis, and it is to the attainment of early and correct diagnosis that those who are investigating the subject should direct their efforts.

Thirteen years ago, John B. Murphy wrote: "We believe that, in cases of sarcoma, the diagnosis can and should be made entirely by the history and with the aid of *skiagrams*." Today this truth enunciated by Murphy still stands. The clinical history and the radiogram are the foundation stones of the diagnosis. More than half the errors of diagnosis and two-thirds of the delay in making a correct diagnosis are due to inadequate attention to the history of the case, to incomplete examination of patient, and to delay in obtaining skiagrams, or their incorrect interpretation.

In regard to the clinical history, I would emphasize the importance of one factor in the

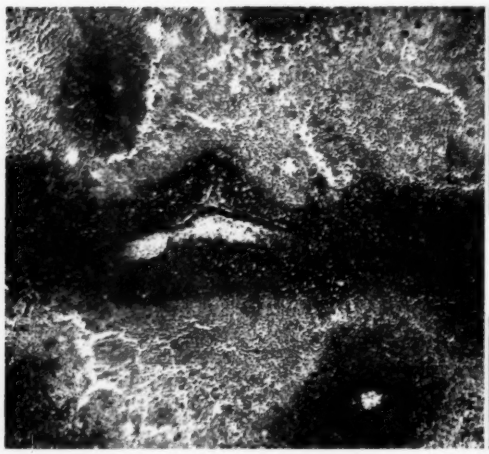

Fig. 4. Typical arrangement of tumor cells around blood vessels (low magnification). Section of tumor (Fig. 3).

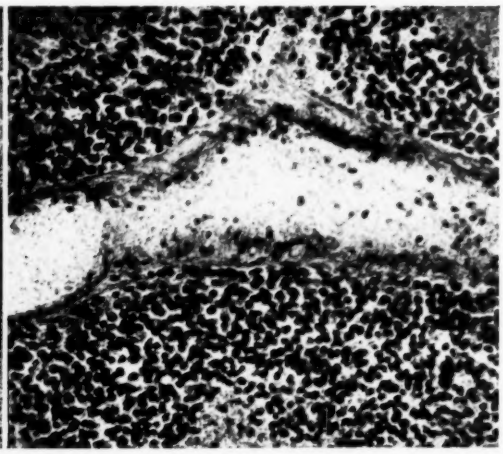

Fig. 5. High magnification of the same section as that shown in Figure 4.

causation, a factor which the histories reveal with great frequency and which has been the subject of much discussion, that is, trauma.

Anyone with even moderate clinical experience must have been struck by the frequency with which a history of trauma is found in the cases of bone tumor. Even if one discards everything doubtful or inconsistent which might mislead him, still there remain a number of facts so suggestive in their evidence as to convince one of the truth and importance of this factor as a cause of such tumors. No writer, among those who have dealt with ample statistics, from Gross to Meyerding, from Sebestyen to Coley, has failed to give due consideration to this important phenomenon. In our own statistics on malignant tumors, trauma is recorded in 47.16 per cent of the cases. It should be understood that if the connection between trauma and tumor is accepted it must be based on indisputable fact. In this connection I accept the premises laid down by Segond long ago and better defined by the results of recent research, which demonstrate that isolated, direct trauma usually induces a tumor of the peripheral layers of the bone with a short latent period; whereas indirect trauma, open wounds, distortions, and fractures, are causes of sarcomata with a long latent period and a central situation.

SYMPTOMS

No specific symptoms are associated with malignant bone tumors. In my report to the Società Ita'iana di Ortopedia, 1927, I said, that if one wishes to discover an osteogenic sarcoma at its origin one ought to live constantly on the lookout for it. One's suspicion may be confirmed by a skiagram, in which case one will have succeeded in diagnosing a tumor of which there was no suggestive sign, except those common to all ordinary lesions.

It has been said that pain is one of the most common and most characteristic signs of a neoplasm. I think that this is an exaggeration, for one can never attach to a subjective phenomenon like pain the importance which we associate with a pathognomonic symptom. More than pain itself, it is the *peculiar quality of the pain* which should make us think of sarcoma, that is a pain which is intense, dull, constant, and localized but not accounted for by other local signs. But again in Ewing's tumor the intermittent, spasmodic pain which is associated with the rise in temperature is not suggestive of neoplasm but is typical of an inflammatory condition, that is osteomyelitis. On the other hand, one frequently observes that tumors of the pelvis and last lumbar vertebra induce pain which has all the characters of ordinary sciatica.

Fig. 6. Spine metastasis of a cancer of the stomach.

Fig. 7. Border-line giant cell tumor.

In my opinion, importance should be attached, even more than to pain, to the question of the *location of the tumor:* a tumor of the diaphysis is almost always an osteogenic sarcoma of the periosteal type, or it is an endothelioma; a tumor of the metaphysis, if it is not innocent, is a sarcoma of the central type; an epiphyseal tumor, in the great majority of cases, is a giant-cell tumor. Tumors of the astragalus and os calcis are either giant-cell tumors or else myxomata. A localized tumor of the vertebral column, unless it is secondary, is either an angio-endothelioma or else a giant-cell tumor.

The location of the growth, therefore, only enables us to distinguish one type of tumor from another. The differential diagnosis, which is most important for the surgeon, is not to determine the type of tumor, but the much more difficult task to decide whether the condition is due to a tumor or an inflammatory disease; also whether the tumor is primary or secondary. Diseases of a definitely infectious nature, such as osteomyelitis, syphilis, and tuberculosis; also dystrophic diseases, both localized and diffuse like Paget's disease; and osteitis fibrosa, all these may simulate a tumor, just as an endothelioma may take on the aspect of osteomyelitis. And how many tumors that we persist in regarding as primary are possibly none other than isolated metastases of visceral neoplasms. The history, the progress of the case, the physical examination, and biological tests may give us valuable information toward the differentiation, and may sometimes by themselves alone decide the diagnosis, but in such cases the last word will always remain with the radiologist.

I do not believe this last point can be disputed. Even the pathologists are convinced of this truth. At the present day, the surgeon can be helped in his diagnosis only by those few pathologists who are equally competent to interpret the histological preparation and the X-ray picture. The great authority on this subject which Codman and Ewing have acquired comes from their vast experience in both pathology and radiography. In a report to the Congress on Cancer held last July in London, Ewing said: "The great majority of

Fig. 8. Metastatic tumor of the head of the humerus developed 10 years after operation for tumor of the thyroid.

bone sarcomata can be detected and the classification made with reasonable accuracy on radiographic signs." We may today leave unopened and ignored all pathological treatises in which the chapter on tumors of bone is treated by authors who are not expert in reading X-ray pictures.

But is it true that definite signs exist which enable us in every case to distinguish, by means of X-ray examination, a tumor from an inflammatory process, or a primary tumor from a secondary one? Certainly not! The X-ray signs are only relatively specific. A bone reacts against a tumor in the same way that it does to destructive stimuli which are non-neoplastic, or even to regenerative processes, as for example in the callus of a fracture. If we delude ourselves by picking out X-ray signs as pathognomonic, it is because we have more or less learned to judge of their relative value, that is, of the comparative value of certain manifestations. Is it not by an analogous process that we judge the so-called specificity of the cells in making the histological diagnosis of tumors? In spite of this difficulty, comparative analysis has reached a stage of accuracy which allows us to gain from a study of the X-ray findings diagnostic information of capital importance. No other method of investigation enables us to recognize in so rapid and comprehensive a way the anatomical features of the tumor, that is, its situation, extent, structure, and relations. Of course, one must not overestimate the importance of this method of investigation. One must not demand of it more than it is able to give. Although in certain cases it alone suffices for the diagnosis, yet in many others it forms only one part of it. But one can never insist too strongly that much more frequent and extensive use should be made of the X-ray than is usual.

It is discouraging to see that although the average surgeon always demands a roentgenogram of a fracture, which could probably be quite well recognized and treated without one, yet very few feel it necessary to make a roentgenogram of the seat of an unexplained pain, although this is often the first and only symptom of a sarcoma.

The stress which we lay on the X-ray in the diagnosis of sarcomata of bone, does not imply that we fail to appreciate the value which biopsy always has had and will have. On the contrary, we are absolutely convinced that it always constitutes the final court of appeal. But owing to circumstances, it is rare that biopsy can be of real value to the surgeon or patient, because, firstly, it is always difficult, often impossible, even for the most experienced pathologist, to interpret the structure and clinical significance of bone tumors and of the diseases which have affinity with them.

An expert pathologist like Dr. MacCarty, of the Mayo Clinic, has said justly: "The differentiation of pathological conditions and their clinical interpretation in the light of the best interests of the patient is an art." How many pathologists at the present day possess this art in the wide and difficult field of diseases of the skeleton?

Few indeed, I believe!

But it is not entirely the pathologist's fault. One must acknowledge that the pathologist is rarely in a position to make a correct and satisfactory decision. Osteosarcomata, as also some giant-cell tumors, never consist of a homogeneous mass, as the epithelial tumors

usually do, but of a conglomeration of diverse elements, scattered in disorderly fashion, so that the structure of the tumor in one place may be totally different from that in another. For a correct diagnosis, it is essential to have a complete examination of all parts of the tumors, which is seldom possible, is even more rarely carried out, and often results in loss of precious time. By having neglected to examine completely a tumor, which I regarded as benign, I lost a patient whom I might have saved by an amputation. And what shall we say of the efficacy of those partial examinations, which are so common, and in which the pathologist is compelled to decide on the nature of the disease from a fragment of tissue, which can reveal only a limited aspect of it? I am not among those who reject biopsy on account of the damage it may do, damage which can be avoided by correct technique, but among those who reject it because I believe that in the great majority of cases biopsy is found to be inadequate. Moreover, this skepticism over the real value of biopsy, which is felt by many surgeons, is shared by a pathologist like Ewing, who is rightly considered an authority on the diagnosis of bone tumors.

I should like to linger in my analysis of the diagnostic problems of bone tumors, so great is the importance that I attach to them, but I must profit by the short time which yet remains to me in order to consider some other aspects of the subject.

CLASSIFICATION

Hitherto classifications of bone tumors have been based exclusively on histological considerations. But the X-ray, in helping us to follow the whole natural history of the tumor, and thus giving it a more complete entity, has produced a revolution in the field of the clinical pathology of bone tumors, and so necessitated a revision of the old classifications. To the Committee of the Registry of Bone Sarcoma is due the credit for carrying out this revision. Whatever may be one's opinion of the classification suggested by the Registry, to my mind it has fundamental value, on the score that it is the first attempt at classification in which, thanks to the happy collaboration of surgeons and pathologists,

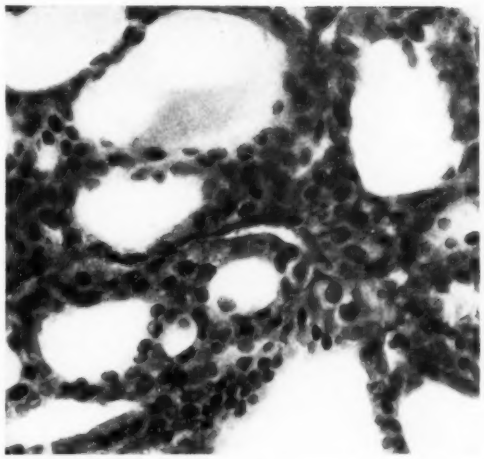

Fig. 9. Section taken from the tumor represented in Figure 8.

weight has been given to clinical and X-ray, as well as to histological criteria. I do not agree entirely with this classification in respect to the terminology and the order of the various groups. I think that certain tumors, for example the angiomata, do not deserve to be placed in a distinct group. On the whole I prefer the classification followed by Nové-Josserand and Tavernier, a classification which certainly was formed under the influence of that of the American Registry, and I agree with the more schematic one shown by Ewing at the recent Cancer Congress. None the less the attempt made by the American Registry is worthy of the greatest praise, because it has cleared the field of conceptions that no longer accorded with facts, and also because it has re-awakened interest and discussion over a subject which had fallen into oblivion.

Endothelioma. A place in modern classification is given to a family of tumors, that of the endotheliomata, which were not previously noted, and this innovation has led to much debate. To some authors, such as Lecène, Masson, Ribbert, these tumors do not seem to form an oncological entity; to others, such as Delbet, they only represent metastases from primary gland tumors. I have no time to enter into the merits of this question, but as the

Fig. 10. Bone metastasis (osteogenic variety) of a cancer of the prostate.

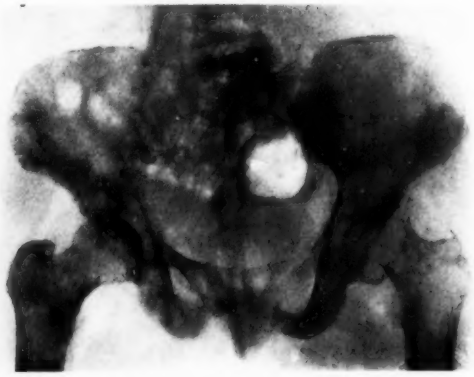

Fig. 11. Bone metastasis (osteolytic variety) of a cancer of the breast.

result of my clinical experience I feel bound to say that such a distinction is actually confirmed by facts, certainly as regards that endothelioma which Ewing has defined by the name of diffuse endothelioma or endothelial myeloma.

It is still a matter for discussion whether this tumor is formed, as Ewing believes, from the perivascular endothelium, or from endothelial cells of bone marrow; or whether, as Kolodny and more recently Oberling have asserted, it arises from the reticulo-endothelial system; whether it belongs to the neoplastic diseases, or whether it ought to be included under those diseases which are now called pretumoral or paratumoral; but what is beyond dispute is that the endothelial myeloma has its own peculiar characteristic, that is to say, clinical, radiological, and structural peculiarities, which enable it to be distinguished from other tumors and from non-neoplastic diseases of bone. Anyone who has observed even one of these cases can have no doubt on the matter, and cannot fail to recognize that this discovery has thrown new light on the obscure pathology of bone tumors. From the moment that the theoretical existence of this form of tumor was announced, there have been published an increasing number of cases, which have confirmed the fact of its existence, while observations multiply showing the diagnostic errors due to the very close resemblance between endothelioma and certain inflammatory processes of bone, more particularly osteomyelitis.

But is Ewing's tumor a primary tumor? This seems to me at present the more important question and most difficult to settle. Its extreme sensitiveness to the X-ray would make one regard it as a secondary tumor, for it is known that the metastases of a bone tumor have a similar sensitiveness. In one case in my series, neoplastic foci appearing first in one tibia and then in the other and in the femur, preceded shortly the appearance of a tumor which occupied the posterior mediastinum and which showed the same sensitiveness to X-rays as did the bone foci. This roused my suspicion that these foci were only metastases from a primary tumor situated in the mediastinum.

Secondary tumors. Therefore, I believe that it is not improbable that we shall be better informed about the origin of endotheliomyeloma when we have gathered more information about the frequency and the ways of distribution and of evolution of secondary tumors. It has long been known that gland tumors, more especially the mammary, thyroid, prostate, and suprarenals, frequently give metastases in bone. But that there exist carcinomatous and sarcomatous tumors of bone, which have originated from visceral blastomata, which cannot be diagnosed clinically, is a more recent discovery, which is daily gaining wider acceptance. This subject

was dealt with at length by Prof. Alessandri in your Congress of 1926. In his recent report to the American Orthopedic Association, Coley expresses surprise that in my statistics there is such a striking number of secondary tumors; but the reason is that my experience with many cases has taught me to classify as secondary tumors those localized and diffuse lesions of bone the nature of which remained uncertain only because the primary tumor was not to be found on clinical examination, but was discovered at the postmortem examination.

In other words, the idea that our conception of secondary neoplasms must be widened, is one which is daily gaining ground. Thus alone can one explain phenomena which otherwise seem inexplicable, and thus we may perhaps one day make clear the reasons for the structural relationship which exists between certain neoplastic bone syndromes and others which hitherto have been considered as inflammatory or as dystrophic, and which perhaps we ought to accustom ourselves to class as pretumoral or paratumoral.

TREATMENT

And now one word as to treatment.

I have had no experience with radium, and a very limited one with Coley's toxins. I have used radiotherapy in association with surgical treatment, and also have utilized it as the only method in dealing with inoperable tumors. I have observed the great value of radiation for the relief of pain, especially in diffuse sarcomata. I have noted the extreme sensitiveness of soft sarcomata, more especially the endotheliomata, for which a few doses suffice to clear away all external evidence of the tumor. But this disappearance is only temporary. Indeed, I have gained the impression that, in some cases, radiation hastened the formation of metastases. Perhaps, in view of their great sensitiveness to X-rays, these tumors could be cured if they were treated early with intensive doses and a wide field of exposure.

In osteogenic sarcoma, I have had some rare successes with tumors of the fibromatous type, but no cure with the periosteal, subperiosteal, or teleangectatic.

I have had no experience of radiotherapy in giant-cell tumors, which I have preferred to treat up till now by surgery in the form of curettage or excision.

In regard to these latter tumors, I may say that I also have observed with what relative frequency relapse occurs after curettage, and how, though rarely, they may produce metastases. What Kolodny has said in this connection is certainly true, namely, that these tumors are benign in the oncological sense of the word, but clinically they may offer very serious problems to the surgeon. Possibly surgical treatment is not the best method of handling these tumors, the tissues of which are so sensitive to mechanical stimuli. It seems logical, as proposed by Ewing, to treat them in the future with radiation alone.

Surgical intervention is still our sheet anchor in the treatment of osteogenic tumors. The few successes which my statistics show represent cases treated with amputation or disarticulation. As I have no means of judging how much radiation immediately after the amputation contributed to the success, I cannot give definite reasons for the results. They are evidently influenced by anatomical, biological, and even accidental conditions, which escape us. Still it is logical to suppose that an early intervention will be more successful than a late one, but early treatment is rarely practicable. In most cases, the tumor is recognized as such only when it is fully developed, or if it is recognized early, the patient very often refuses amputation.

No patient hesitates to undergo resection of the stomach or intestines on the mere supposition of a cancer; but one must work hard to convince a patient with extensive osteosarcoma of the necessity of an amputation.

Uncertainty in the diagnosis, hesitation on the part of the patient, reluctance of the surgeon to undertake a mutilating operation, inevitable and almost always useless attempts to cure by means of conservative measures, cause us to lose precious time and take from the radical operation most of its chances of success.

The results of the treatment of bone sarcoma are certainly discouraging. In our present state of knowledge, our own means of

improving them is by making better use of the means at our disposal.

It is much to be hoped that X-ray therapy has not yet exhausted its resources in the treatment of bone sarcoma. As for operative treatment, not much can be expected from the perfecting of technique; its efficiency will increase step by step as we learn to discover the first manifestations of tumors and to operate without delay.

I hope you will excuse me for having merely touched on a few points in the complex subject which I chose, but in order to say much more, I should have had to abuse your kindness too much.

I shall have attained the end I desired, if I have succeeded in fixing your attention on a problem of great scientific and practical interest.

Certainly, in the study of tumors we have reached the dead point, which we can pass only on the day when we shall be better informed about their intimate nature. By what routes we shall reach the discovery of the causes of tumors it is impossible to foresee, but one thing is certain, and that is that this discovery neither will be the fruit of chance, nor will it be a miracle, but it will be the logical conclusion and reward of hard work and intensive research.

Let us, therefore, be inspired by the example of John B. Murphy and follow tenaciously the road which shall lead us to the desired goal.

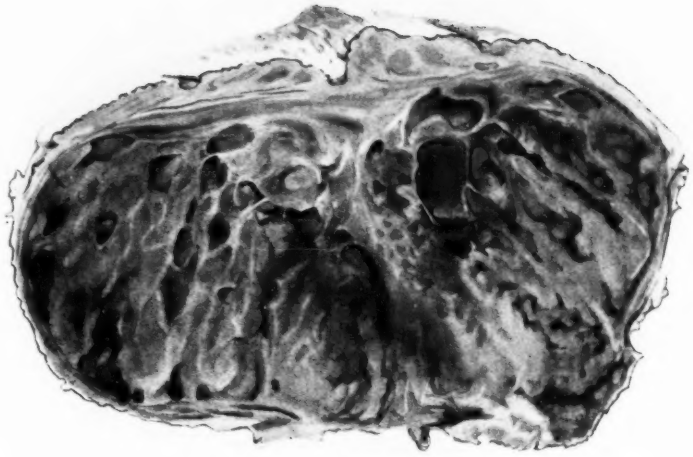

Fig. 1 (at left). Sacrococcygeal chordoma. The original tumor showing the dimple at site of puncture 6 months before. (Case 1.)

Fig. 3. Water-color drawing of a section of the primary tumor, showing the skin surface above, the extensive hyaline degeneration of the stroma, and many areas of hæmorrhage. (Case 1.)

Chordoma—Andrew J. Hutton and Archibald Young.

CHORDOMA

A Report of Two Cases: A Malignant Sacrococcygeal Chordoma and a Chordoma of the Dorsal Spine[1]

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AND
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ONE of our purposes in offering this short communication to the Clinical Congress of the American College of Surgeons is to remove the reproach of having the name of one of us included in the now considerable literature on the subject of chordoma against a still "unpublished observation." The case which we have to report first is included in the bibliography attached to the paper on "Chordoma" published in the January, 1926, number of *The Journal of Pathology and Bacteriology*, by Professor Matthew J. Stewart (Leeds) and Dr. J. E. Morin (Quebec). It appears there as an "unpublished observation" against the names of A. Young and R. Muir. The specimens were shown by Professor Muir at a meeting of the Pathological Society of Great Britain and Ireland, January, 1925.

In publishing now a full record of the case, we are able to record its later history, and to describe the condition when a somewhat late recurrence called for further operative intervention.

We take the opportunity of including, along with the report of the earlier case, the account of a second with which we have had to deal more recently, which has the additional interest that the region of the spine involved, namely, the dorsal spine, has not hitherto been found to be the seat of chordoma; at least we have been able to find, in the literature, no record of a similar case.

This second case has been included already in a paper by Dr. D. F. Cappell, of the pathological department of the University of Glasgow, to be published in *The Journal of Pathology and Bacteriology* for October, 1928, along with accounts of two other cases of chordoma affecting the vertebral column, namely, of the cervical spine. We report the first of these cases here independently, but we desire to acknowledge our indebtedness to Dr. Cappell for the description of the histological findings and for his interest in the case.

In choosing the venue for our contribution, we have been influenced by the fact that American surgical literature, though it contains several of the earlier examples of the condition, is singularly lacking in recent records of chordoma.

CASE OF MALIGNANT CHORDOMA OF THE SACROCOCCYGEAL REGION

N. G., a male, aged 49 years, was admitted to the Western Infirmary, Glasgow, on May 11, 1924, with a very large tumor bulging from his sacral region. He was sent in by Dr. John Miller, of Greenock, from whose letter the following may be quoted:

"The condition extends over several years, and an operation was performed initially, when the report stated that tissue removed was inflammatory. The tumor rapidly grew, and 6 months ago it was certainly the size of an adult's head. Prolonged X-ray applications have reduced it to its present size. When it was seen 6 months ago, I inserted an exploring needle, with a resulting hæmorrhage which lasted for several days. The tumor at the time was very soft, suggesting fluctuation. With its decrease in size, it has become very hard. My view is that the condition is a sarcoma."

The particulars given by Dr. Miller may be amplified. Inquiry elicited the fact that, 20 years before, at the age of 29 years, the man fell heavily on his buttocks. It was not, however, until 1920, when he was 45 years of age, that anything out of the normal was noticed. About that time, that is, 4 years before admission, pain of a dull type began to be experienced in the sacral region, and within 6 months of the first appearance of the pain, the onset of a swelling was observed. This swelling grew with some rapidity until it reached the size of a cocoanut. Some months afterward, in the Greenock Infirmary, the tumor was incised, and material was removed for histological examination. This examination, made by a Clinical Research Institute, resulted in a report that the tissue was of an inflammatory nature. Only a portion of the tumor was

[1] Presented before the Clinical Congress of American College of Surgeons, Boston, October 11, 1928.

Fig. 2. At left, photograph of recurrent tumor 4 years after. At right, same patient showing the superficial extent and bulk of the tumor and the scar of the former operation below.

removed, and the growth of the mass continued steadily to increase. Later, under the influence of prolonged X-ray treatment, considerable retrogression of the tumor is said to have taken place, and, as indicated in Dr. Miller's letter, the tumor was explored with the result that alarming hæmorrhage took place, which continued for some days. Again there followed a certain diminution in size and a change from the soft, almost fluctuant consistence to one considerably harder. The exploratory puncture was carried out in November, 1923, and X-ray treatment was resumed afterward in the Greenock Infirmary, with some further diminution in size.

Fig. 4. Photograph of same section as shown in Figure 3 (insert).

Condition on admission to the Western Infirmary, Glasgow, in May, 1924. The tumor, which was fully the size of a child's head, was situated in the sacral region, bulging more to the left than to the right. It was very little movable on the sacrum, and the skin over its surface was somewhat closely attached to it. At one spot there was a deep dimple, almost suggesting in appearance and site the post-anal dimple of congenital origin. The dimple, however, represented the result of the puncture which had been made by the exploring needle 6 months before (Fig. 1, insert).

The tumor was hard throughout, though varying in degree of hardness at different places. There was no suggestion of fluctuation at any place. Apart from the bulk of the tumor, and the inconvenience directly traceable to this, there were no marked symptoms of any kind, beyond occasional slight pains down both lower limbs, and an indefinite sensation of ineffective defæcation. Rectal examination showed that the tumor bulged also into the pelvic cavity, and that it had a close relation with the wall of the lower rectum. At no point, however, was there any indication of actual involvement of the rectal wall in the growth, and the palpating finger seemed to be able to move the mucosa freely upon the mass of the tumor outside it. The man had marked varicose veins in both legs, and the scars of old varicose ulcers on the left leg.

X-ray examination of the tumor and of the lumbosacral spine, at this time, was somewhat inconclusive. The lower end of the sacrum was not easily distinguishable from the tumor. The outline of the coccyx was quite indefinite. It was thought that

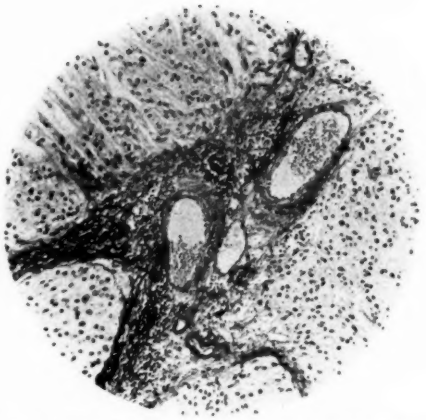

Fig. 5. Showing the stroma between several adjacent alveoli, the margins of which are seen. The blood vessels have rather thin walls, and the connective tissue is infiltrated by round cells and by phagocytes containing altered blood pigment. The peripheral cells in the alveoli are well preserved and are seen to form long strands with an irregularly radiate arrangement. (Case 1.)

Fig. 6. Margins of the larger alveoli, which have been stained to show mucin. It will be noted that the strands of tumor cells are separated by varying amounts of intercellular mucoid matrix which, in places, forms large masses in which tumor cells have disappeared and that the stroma has here undergone hyaline degeneration. (Case 1.)

there was some irregular bone formation in the region of the second and third lumbar vertebræ, particularly the lower aspect of the second lumbar.

Operation, June 6, 1924. A long, slightly curved incision was made across the surface of the tumor, with the convexity of the curve upward—a segment of the overlying skin being removed subsequently along with the tumor. By careful and laborious dissection the tumor was freed from skin, from the levator ani muscle, and coccygeus, and in front, with considerable difficulty, from the wall of the lower rectum. So closely was it attached to the wall of the rectum that a portion of the latter seemed to be endangered. Above, it was not found possible to separate the tumor cleanly from the coccyx or lower end of the sacrum, which had to be cut across with bone-forceps; coccyx was almost entirely destroyed, and tip of the sacrum was taken away with the tumor mass. After removal of the tumor, the lower rectum bulged into the space left, and it had to be supported by plastic suture of levator ani muscles and fascia, so as to endeavor to form a secure pelvic floor. The wound was closed in layers, provision being made for drainage.

Immediate after-course. In spite of the somewhat extensive operation, and the large wound which could be closed only in part, healing took place practically *per primam.* There was very little difficulty with the bladder or bowels, and the patient was dismissed from hospital a month after operation, apparently well. There was no sign of tumor, and, except for a subjective sensation of weakness in expulsive power at defæcation, due to the weakened pelvic floor, the man seemed little the worse for what had been done.

Later course. The man reported himself at intervals during the following years, up to January 5, 1927, during which period—two and a half years—there was no sign of recurrence. At each visit the man looked well, and expressed himself as feeling well. From January, 1927, until April, 1928, he failed to report, but on the second of these dates he presented himself again with unmistakable evidence

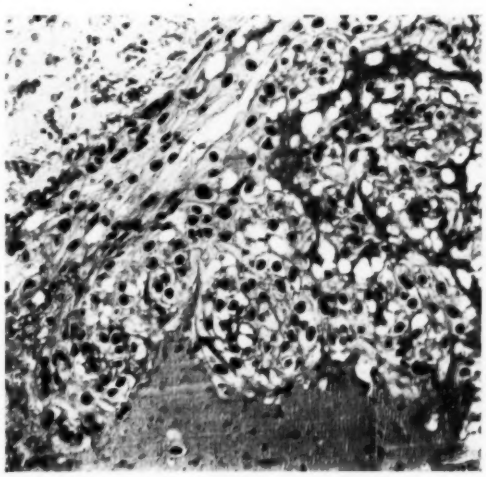

Fig. 7. High power photomicrograph showing highly vacuolated tumor cells, with a moderate amount of intercellular matrix. (Case 1.)

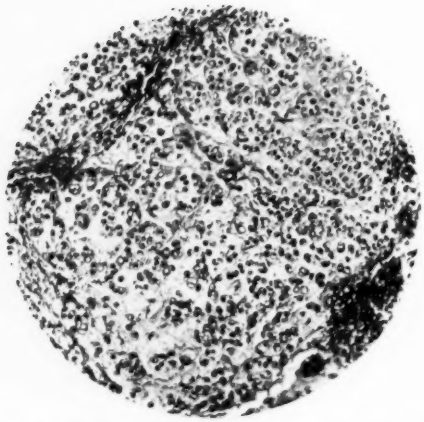

Fig. 8. Photomicrograph which shows scanty stroma and the presence of hæmorrhage. The arrangement of the cells bears a distinct resemblance to cartilage. (Case 2.) ×70.

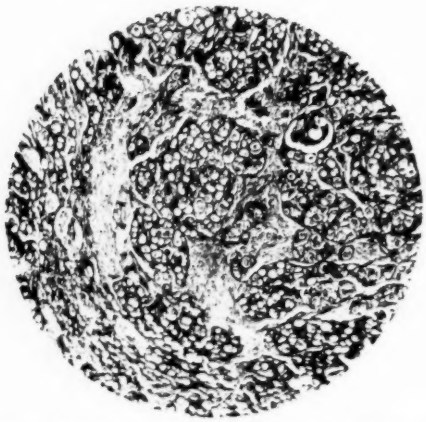

Fig. 9. Photomicrograph of similar field to that shown in Figure 8, stained to show mucin. The nodular character of the growth is well seen, and the condensation of the matrix around each cell is brought out. (Case 2.) ×70.

of renewed tumor growth in the region of the original site.

He gave the story that he had felt nothing wrong until May, 1927, i.e., 4 months after his last previous visit, and practically 3 years after his operation. At this time he became conscious of some swelling about the site of the original tumor, but it was not until 4 or 5 months later that the swelling became at all marked. It did not trouble him much at first beyond a slight dull ache. Later, however, pain became more pronounced, was occasionally sharp in character, and radiated up into the back but not down into the legs. For a time he was troubled with diarrhœa, but this passed off, and was replaced by increasing difficulty in obtaining a satisfactory bowel movement, unless after the use of laxative medicine. Liquid paraffin medication proved quite effective in overcoming the difficulty, which seemed to be largely a mechanical one. He sometimes felt sick, but did not vomit. A few months before he presented himself in April, 1928, he began to have trouble with micturition,—chiefly delay in initiating and in completing the act,—but there were no frequency and no pain associated with it. While the tumor was the seat of pain when he moved about, he had no pain in his legs, nor had there been any swelling of the latter.

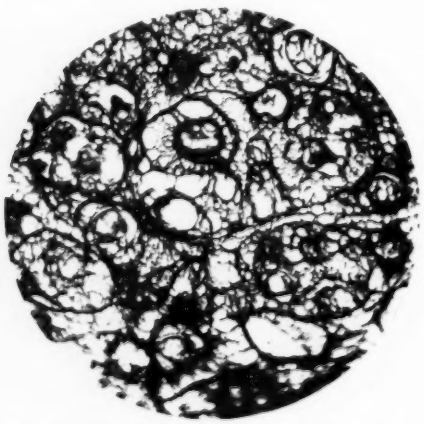

Fig. 10. High power photomicrograph showing a large physaliphorous cell toward the center of the field. (Case 2.) ×350.

Condition at time of re-admission to hospital on May 5, 1928. At the time of his re-admission to hospital, there was an obvious tumor in the left sacral region, estimated roughly as about the size of a large grape-fruit. It was of somewhat ovoid shape. The skin could be moved over it, but the tumor was closely attached on its deep aspect. It was firm and non-fluctuant, and a number of markedly engorged veins coursed over its surface. The scar of the previous operation was situated over the lower medial pole of the tumor, and at one spot, about the middle of the scar, the skin was reddened, suggesting the approach of tumor growth toward the surface. At this spot the tumor felt a little softer than elsewhere (Fig. 2).

Rectal examination showed that the mass bulged considerably into the pelvis, as the original tumor had done. It could be felt compressing and stretching the rectal wall, which was not readily moved over it by the palpating finger. The mucosa, however, was intact, and could be moved over it.

Operation, May 22, 1928. By reason of the close incorporation of the tumor with the muscular floor

of the pelvis, and its close relation to the lower rectum, as well as its apparent continuity with the lower sacrum, this operation was difficult and laborious.

The superficial part of the tumor was first of all cleared from the overlying skin, a part of the old scar being removed with the tumor. The deeper part of the tumor had to be separated very carefully from the rectal wall, but this was successfully accomplished without opening into the lumen of the bowel, or apparently damaging its wall. At the upper part of the tumor, it was found to be continuous with the sacrum, which was invaded by tumor growth. An endeavor was made to get beyond the growth, by removal with it of a further portion of the sacrum, but in the process the tumor was unavoidably opened into. Very thorough scraping away, of the invading tumor tissue, and of the obviously invaded bone, was carried out, but it could not be said with confidence that complete removal was satisfactorily effected. Accordingly, after the pelvic floor had been reconstituted, and the wound had been for the most part closed, the area under suspicion was surrounded by a barrage of radium, introduced in a ring of tubes which enclosed the lower segment of the remaining sacrum. These radium tubes were left *in situ* for 36 hours.

After-course. The immediate healing of the wound was uneventful as before, though, probably as the result of the radium, the extreme upper end of the wound was a little slow in closing. There was never, however, more than a little thin serous discharge. The parts ultimately cicatrized firmly, and the man was dismissed from hospital on July 7, i.e., 6½ weeks after the operation, with the wound completely healed. He seemed well, and was free from pain. His only discomfort was a feeling of futility or ineffectiveness when he strained in the effort to pass urine or to defæcate. This was due to the inevitable weakening of the pelvic floor, as the result of the two operations. By the use of simple laxatives, however, quite good evacuations of the bowels could be obtained without much trouble. So far as urination was concerned, the man had gradually acquired, with practice, fair facility in emptying his bladder by the avoidance of any effort at straining. It is too soon yet to attempt to form an opinion as to the further prognosis in the case. Examined before his dismissal from hospital, no sign could be made out of any fresh growth, either externally or palpable from the rectum.

Pathology of the tumor. Sections both of the primary tumor and of the recurrent tumor were found to conform generally with the histological characters of a typical chordoma, to which reference will be made later. Particularly, it was found that the tendency of the cells of the alveolar masses to mucoid degeneration was well marked, abundance of mucin droplets being present both intracellularly and between the cells.

There was extensive hyaline degeneration of the supporting fibrous stroma, and areas of hæmorrhage were present throughout the tumor in considerable number. Many of these areas were of some size, especially in the center of the larger lobules. The photograph of the patient and the water-color drawing of a mesial section of the primary tumor (Figs. 3, insert, and 4), illustrate very well both the areas of hæmorrhage and the degenerated stroma.

Histology (Dr. Cappell). We are indebted to Dr. D. F. Cappell for the following detailed description of the histological findings both in the primary tumor and in the material from the second operation. Dr. Cappell's report is embodied herewith (Figs. 5, 6 and 7).

Primary Tumor: "The tumor exhibits a frankly alveolar structure, with widespread mucoid degeneration of the cells. The growth is surrounded by a dense fibrous tissue capsule, which in many places is in a condition of hyaline degeneration.

"The stroma which separates the alveoli of tumor cells carries fairly numerous blood vessels, often with rather thin walls, so that hæmorrhage into the degenerate parts of the tumor is common. Numerous lymphocytes, and phagocytes containing altered blood pigment, are present in the stroma, which is often very hyaline.

"The tumor cells are best preserved at the alveolar margins, but are found to exhibit extreme polymorphism in different parts of the growth. There are areas of small polygonal cells with rather empty cytoplasm and round or oval nuclei.

"At an early stage, these cells begin to produce a mucinous secretion, which at first accumulates within the cells, producing a vacuolated appearance. This secretion is generally poured out into the intercellular spaces, and leads to the characteristic separation of the tumor cells into irregular trabeculæ, which tend to assume an irregularly radiate arrangement. The mucoid secretion accumulates toward the center of the nodule of tumor cells, and leads eventually to the formation of large masses of mucoid material in which the surviving cells are situated only at the periphery. Into such masses hæmorrhage often occurs. The secretion does not always escape readily from the cells but accumulates within the cytoplasm, producing large vacuolated cells—the so-called physaliphorous cells. Many of these cells exhibit nuclear hyperchromatism, and in places multinucleated syncytial giant cells are formed. The nuclei of the tumor cells are often markedly vacuolated, and actual physaliphorous nuclei are readily found.

"In the material from the second operation, the tumor presents a structure almost identical with that removed previously, but irregularity in size and shape of the cells is rather more pronounced, and multinucleated syncytial giant cells, with hyperchromatic nuclei, are more abundant.

"The stroma in the more recent material is often overrun with polymorphonuclears, but this is probably attributable to the influence of necrosis and degeneration."

CASE OF CHORDOMA OF THE DORSAL SPINE

A. D., a male, aged 55 years, was admitted to the Western Infirmary, Glasgow, on September 21, 1927, under the care of Dr. R. Barclay Ness. His complaint was of loss of power in the lower limbs, which had developed gradually during the 9 months immediately preceding.

History. The first symptom observed was that the legs got easily tired, the right being more affected than the left from the very beginning. Soon after the onset there was found to be some difficulty in co-ordination. He staggered and swayed in walking. Along with the development of this inco-ordination, he had pain and a sense of constriction around the lower part of the chest. This, however, had largely passed off before his admission to hospital. For 3 weeks before his admission patient had been confined to bed almost altogether. When, occasionally, he did get up, he had the sensation of walking on velvet. Subjectively, he felt his legs and feet cold and numb from the knees down. His previous health had been good, and his family history presented no point of special interest.

Summary of condition while under investigation in medical ward. The patient was a fairly well developed man of rather spare habit. He had a slight degree of scoliosis to the right in the lower thoracic region. There was no evidence of impaired function of the cranial nerves, or of any of the nerves of the upper limbs. The abdominal reflexes were absent. There was at first no incontinence of bladder or bowels, but there was some delay in micturition. Later, there was, for a considerable period, definite lack of control of the bladder (which continued for a time even after his operation). There was no pain in the lower limbs during his residence in hospital.

Loss of power in the lower limbs was very marked, especially in the right. It involved the whole limb from the hip downward. He was quite unable to stand alone. There was no tremor, and at first the knee and ankle jerks were not exaggerated. Later they became much exaggerated. There was a positive Babinski sign on both sides from the time of admission, and ankle clonus could be elicited on the left side, but not on the right.

Subjectively, both legs and feet felt numb, and there was defective sensibility for temperature and touch, reaching as high as the nipple line on both sides. The muscle-joint sense was unimpaired.

The Wassermann test, both blood and cerebrospinal fluid, was negative, and lumbar puncture yielded at first a clear cerebrospinal fluid of normal character, at a pressure of 100 millimeters of water. The pressure response was normal, but there was a slight increase on coughing.

X-ray examination of the spine revealed a definite abnormality of the fifth and sixth dorsal vertebræ, the intervening disc being apparently destroyed and merged in an ill-defined mass, which involved both the contiguous vertebral bodies. The discs above and below these vertebræ were thinned.

Further progress while in medical ward. During the 3 months he remained in the medical ward his condition got steadily worse. Spasm of the lower limbs became a very marked and distressing feature. Bed-sores formed over the sacrum, over the trochanters, and elsewhere, on the slightest provocation. These sores healed only with great difficulty and very slowly. They readily broke down again. A later examination of the cerebrospinal fluid gave evidence of definite spinal block.

Transferred to a surgical ward on December 14, 1927, with a diagnosis of compression paraplegia due to tumor growth, the man's condition had become much worse. Bladder and bowel control were largely lost, and spasm of the lower limbs was a very marked feature of the condition. Spasmodic flexion and contracture were well developed, and the spasm of the adductors of the thighs was particularly pronounced. The sores over the sacrum and trochanters were barely healed, and broke down afresh very readily.

Operation, December 27, 1927. The operation was carried out without any general anæsthetic, novocain anæsthesia being employed. The surface tissues for some little way above the region of operation were first of all infiltrated, and thereafter the nerve roots for several segments above were blocked. The result was satisfactory, the man experiencing no pain during the operation.

The spinal column was exposed in the usual manner, and the spinous processes and laminæ were removed from the third to the sixth dorsal vertebræ inclusive. The cord was found compressed and flattened to the left anterior aspect of the spinal canal by a tumor, entirely extrathecal, involving the bodies of the fourth, fifth, and sixth dorsal vertebræ. At this level the cord showed no pulsation. The tumor was of a soft, crumbling character. It seemed to have originated from the bodies of the vertebræ, and to have spread into and partially destroyed the corresponding laminæ.

As much as possible of the tumor was removed, and the cord, relieved of pressure, began to pulsate again. Complete removal of the tumor seemed to be impracticable, by reason of the extent and nature of the involvement of the vertebral bodies. It was recognized that the operation could probably only be of the nature of a decompression procedure. The wound was accordingly closed somewhat loosely, in layers, a drainage tube being inserted at the lower end of the wound, and so fixed there that its deep end reached just down to the upper margin of the bony canal.

Immediate after-course. The operation was well borne, and the wound healed *per primam.* The drainage tube proved to be hardly necessary and was removed early.

The first notable result of the operation was the recovery of bladder and bowel control within a few days. Very soon, too, the recurring spasms of the lower limbs became less troublesome; but for a considerable time there was little difference otherwise

in the lower limbs, either as regards sensibility or in respect of power. The tendency to bed-sore formation continued to be a troublesome feature for some time, and indeed for a good many weeks doubt was felt as to whether the man was not going to get steadily worse and die. Ultimately, however, a definite improvement in his general condition took place, and it was possible to get him out of bed for a time each day. From this point he improved more rapidly, and he was dismissed from hospital on April 13, 1928, i.e., 3½ months after his operation.

Later course. This patient reported himself for examination 6 months after his operation, when his general condition was found to be very good. He still had a definite, but much less marked, contracture of the ham-string group of muscles in both thighs, and there was slight adductor spasm; but the spastic condition of the limbs generally, and more especially of the legs (below the knees), was very much improved. The man could get about comfortably in a bath chair, and was able to assist himself from bed to chair, or from chair to bed, standing on his feet and steadying himself by his arms. He was able to take interest in his garden, and he expressed himself as feeling well pleased with the result of his operation. He had occasional difficulty with micturition, and a tendency to constipation, but there was no incontinence either of bladder or of bowel. In spite of his persisting disability, the man was surprisingly cheerful.

Pathology of the tumor. For the following description of the histological characters of the tumor we are indebted to Dr. D. F. Cappell:

"Microscopically, the tumor is composed of a very abundant matrix and fairly numerous cells which show a tendency to irregular nodular formation. There is rather abundant hæmorrhage in places in the mucoid matrix. Around each cell the matrix appears to be condensed, and the cells are slightly shrunken, thus presenting an appearance closely resembling cartilage (Fig. 8).

"The tumor cells tend to be scattered irregularly throughout the matrix, and do not exhibit the characteristic arrangement of chordoma cells, which generally form long strands, with a tendency to a radial arrangement in the nodules, the center of which is often purely mucoid. The tumor cells are generally round or oval, with a deeply staining nucleus, a well defined nuclear structure, and a single nucleolus, and present all gradations from small cells with deeply staining cell bodies to large vacuolated masses of protoplasm, the typical "physaliphorous" cells (Fig. 10). Nuclear vacuolation is not easily seen, but can be found on careful search.

"The inter-cellular matrix is unusually abundant, and gives the characteristic staining reaction for mucin, with polychrome methylene blue and mucicarmine (Fig. 9).

"The stroma is exceedingly scanty in the fragments examined, and the usual enveloping capsule of the growth has not been found, but this is probably attributable to the paucity of the material at disposal. The stroma consists of little more than capillary vessels, with thin walls, poorly supported by connective tissue fibrils (Fig. 8). Around some of the larger and thicker-walled vessels, there are areas of hyaline material which may represent altered mucoid matrix, as it stains more bluish with the polychrome stain and reddish with van Gieson. Around the groups of cells which form the poorly defined nodules previously described, the condensed matrix stains reddish with van Gieson, and presents a vague fibrillar structure; this supports Peyron's idea that transitions between chordomatous secretions and collagen occur.

"The tumor cells are somewhat more numerous in relation to the fibrous septa, and here they present an aspect more typical of chordomatous growths, forming irregular strands which soon become lost in the abundant matrix. Even here, however, the cells exhibit the tendency to dissociation from one another which has been previously described. Fragments of bone from the laminæ of the vertebræ were also examined, after decalcification, but only scanty traces of invasion by tumor cells were found."

It will be observed that Dr. Cappell, when he wrote the above description, was not so confident as he has since become of the diagnosis of chordoma in this case. We have since discussed the case and the appearances with him, and we have little doubt that he has rightly come to the conclusion that the tumor in this case must be classed as a chordoma.

It should be stated also that the sections have since been submitted to Professor Matthew J. Stewart (Leeds), who is definitely of opinion that the tumor is undoubtedly of chordomatous type.

The type of tumor with which alone it might have been confused was a chondroma undergoing degeneration, and it is true that certain features referred to in the above report at first suggested such a diagnosis to Dr. Cappell, but a more comprehensive view of the appearances would seem to negative such a diagnosis. Dr. Cappell draws attention also to the fact that chondromata of the vertebral column are themselves exceedingly rare.

If the opinion of the surgeons, who have seen the actual growth at operation, and have been able to observe the naked eye characters of the tumor, its consistence, its vascularity, etc., is of value in this connection, we would hazard the opinion very definitely,

for what it is worth, that the tumor had little or nothing in appearance in common with a chondroma, and we believe, therefore, that Dr. Cappell's diagnosis of chordoma, on the basis of the histological findings, may be accepted with confidence.

HISTORICAL RÉSUMÉ

The development of our knowledge of tumors of notochordal origin has taken place within the last 30 years, and more particularly since 1922, when Professor Matthew J. Stewart, of Leeds, reported the first case recognized in Britain. In *The Journal of Pathology and Bacteriology*, vol. xxv, 1922, he gave a full account of the case, and also added an excellent historical summary of cases recorded in the literature up to that time.

From that time, further cases have been reported in Britain and elsewhere, with considerable frequency, so that 4 years later, in a further paper, already referred to, Stewart and Morin were able to record a total number of 57 cases. Dr. D. F. Cappell in his paper, also previously referred to, to be published, in the middle of October, in *The Journal of Pathology and Bacteriology*, brings the number of recorded cases, including the three vertebral cases with which his paper specially deals, to a total of 80. This number, of course, includes the two referred to in our paper.

Professor Stewart's first case was that of a man, aged 65 years, who was operated on by Sir Berkeley Moynihan, in May, 1910. The case is of particular interest in view of its resemblance to our first case. The man had a tumor in the sacrococcygeal region, which had been growing slowly for 8 years. In general outline it was hemispherical, measuring 3 inches each way. Moynihan seems to have removed it without any special difficulty.

The tumor was examined by Professor Stewart at the time, and the opinion was expressed that it was a peculiar type of carcinoma undergoing widespread colloid degeneration. It seemed to be well encapsuled. Its true nature was not then appreciated.

Some 9 years later, however, as Stewart states in his original paper, he was led to review the case afresh, as the result of being shown sections of a chordoma by Professor Peyron of Marseilles. These sections evidently recalled to Professor Stewart's memory certain similar appearances in the tumor in question, which he had examined 9 years before.

The patient was accordingly sought out, and was visited in September, 1920, i.e., 10 years and 4 months after operation, and was found to be still alive. He was apparently in fairly good general health, but he had both a local recurrence and secondary deposits. These were: (1) a small nodule, about the size of a pea, in the middle of the old sacrococcygeal scar; (2) a large sausage-shaped mass in the region of the left buttock, running downward and outward, and measuring 12 inches by 3½ inches; (3) a mass, 8 inches by 4 inches, in the upper dorsal region, over the right scapula.

The mass in the left buttock had appeared 5 or 6 years after his operation, and the tumor in the right scapular region 2 years later. The small local recurrence had not been noticed previously. Pain was complained of along the course of the left great sciatic nerve, and the tumors caused considerable discomfort by reason of their size. Six months later, a further tumor appeared in connection with the upper end of the left femur. The man died in June, 1921, aged 76 years.

It will be observed that in this case a tumor was known to have been present for 8 years before it came to operation, and that the man lived for 11 years afterward, a total duration, therefore, of 19 years.

This illustrates very well the frequently very slow growth of tumors of this type, which, though they may be large, and generally are definitely malignant in character, do not, as a rule, lead to an early fatal issue, either from their direct local effects or from metastases.

In the first of our two cases, the first sign of the tumor was observed in 1920, so that the duration is, up to the present, fully 8 years.

In his original paper, Professor Stewart gave an excellent historical summary of recorded observations up to that time. He

dealt with 26 cases of tumors believed to be of notochordal origin—15 at the clivus Blumenbachii (dorsum sellæ), and 9 in the sacro-coccygeal region. The following short outline is taken from his paper.

Luschka, in 1856, described a soft, lobulated jelly-like mass protruding into the skull from the clivus, and perforating the dura mater. Virchow, in 1857, gave what was perhaps the first good description of the condition. He evidently thought that he was dealing with formations of a cartilaginous nature, whose fundamental substance had undergone softening, and whose parenchyma cells showed vesicular degeneration. He therefore applied the term ecchondrosis physaliphora to the condition. The site of origin was in the neighborhood of the spheno-occipital synchondrosis.

H. Mueller, in 1858, was the first to suggest that these tumors were of notochordal origin. He demonstrated "rests" of notochordal tissue in the basilar cartilage of man and animals, and showed that in the fetus the notochord reaches up to the sella turcica. In the spheno-occipital synchondrosis it remains as a small soft mass, analogous to the nuclei pulposi of the intervertebral discs, which are generally recognized as relics of chordal tissue. Mueller also showed that, in the region of the future spheno-occipital synchondrosis, the notochord has a decided tendency to approach the superior surface of the basilar cartilage.

It seems that Ribbert, who confirmed the observations of Mueller in 1894, was the first to suggest the name "chordoma."

The first recorded cases of sacro-coccygeal chordoma of definite clinical interest in man seem to have been those of Feldmann and Mazzia in 1910.

NOMENCLATURE

Stewart suggested that the small jelly-like nodule, having very limited powers of growth, which is met with occasionally, arising from the middle of the clivus, should be termed "ecchordosis physaliphora spheno-occipitalis." This type of tumor seldom gives rise to any large progressive formation, and is met with usually only as a casual finding in the post-mortem room. It may be regarded rather as a simple notochordal protrusion than as a tumor.

Other less frequent tumors in the same situation, having greater powers of growth and capable of producing definite symptoms, and even of leading to death, must be regarded as genuine neoplasms. To a tumor of this type, Stewart would apply the term "malignant spheno-occipital chordoma."

For similar tumors, springing from or related to the posterior extremity of the notochord, and of the spinal column, he suggested the term "malignant sacro-coccygeal chordoma."

Cappell now records three examples of a malignant chordoma springing from other regions of the spine, two from the cervical region, and one (our second case) from the dorsal region. In his paper, Cappell refers also to a limited number of recent observations of the condition in other situations.

NAKED-EYE CHARACTERS AND HISTOLOGY OF CHORDOMA

The description given by Stewart of the naked-eye characters of the tumor in his first case, and of its histology, is so complete and corresponds so closely with what may be regarded as the typical findings in such tumors, that we venture to summarize his description here:

NAKED-EYE CHARACTERS (STEWART)

The tumor is well encapsuled and is of rounded or lobulated outline. On section, the cut surface presents a lobulated appearance, with dense fibrous tissue of varying width separating the lobules. The latter show mucoid degeneration, often of a very advanced character, and the stroma in those regions is often the seat of marked hyaline degeneration. The appearance presented may suggest very strongly that of a colloid carcinoma. Where the mucoid change is less advanced, the tumor tissue may be firm, granular, and opaque, like a fairly cellular carcinoma. Hæmorrhages, both old and recent, and varying in size, are present as a rule here and there through the substance. Such appearances on the cut surface of a tumor of this kind are well illustrated in photograph and water-color drawing from our first case (Figs. 3 and 4).

HISTOLOGY (STEWART)

The tumor is alveolar in structure, and shows a clean-cut separation through the parenchyma and stroma, the whole encapsuled by a layer of dense fibrous tissue. The alveolar masses vary greatly in size, and, while many of them, especially the larger, show advanced mucoid degeneration, others, especially some of the smaller, are richly cellular. The stroma is in the form of strands of fibrous tissue of varying width, much of it showing hyaline degeneration.

Parenchyma. There are all gradations from active cellular tissue to areas of extreme mucoid change. The cells in the former are distinctly epithelial, set close together, often without intercellular substance; while in the latter they are broken up into little masses in the midst of the mucoid material. Cellular outlines are often indistinct, so that at first sight the mass may suggest a multinucleated syncytium, filled with vacuoles and collections of mucoid material. In the youngest, most active looking areas, the cell margins are more clearly made out, the shape of the cells being irregularly polygonal. There is great variation in size, the larger cells occurring chiefly in the richly cellular areas. In regions where mucoid degeneration is advanced, the cells are mostly small and shrunken, and stain deeply.

The mucoid degeneration, which is one of the most striking features of the tumor, seems to begin at an early stage in the life history of the cell. At first the droplets of the mucin are small and *intra*-cellular, but they soon enlarge, and lead to a high degree of cytoplasmic vacuolation. The mucin escapes and collects intercellularly. The collections of mucin ultimately break up the tumor tissue, first into cords, then into little groups of shrunken tumor cells, the whole appearance suggesting the character of the degenerating notochord in the nuclei pulposi of the intervertebral discs. Occasionally, a cell is ballooned out by a large amount of mucin, as if the cell possessed an unusually strong cell membrane, or a specially condensed peripheral zone of cytoplasm. This is the fully developed "physaliphorous" cell of Virchow.

The nuclei of the tumor cells show great variation in size and considerable variation in shape. The majority are oval or spheroidal, 10 to 15μ in diameter. Others are polymorphous, while, in the most degenerate parts of the growth, they may be shrunken, crenated, and very irregular in outline. Nuclear staining varies in intensity. Large, round, hyperchromatic nuclei, 20 to 35μ in diameter, are fairly frequent, chiefly in the more cellular, less degenerate parts of the tumor, where also multinucleated cells may be found. Each nucleus contains one or two, and sometimes three, nucleoli. Mitotic figures are few in number.

Widespread nuclear vacuolation is a striking feature. The vacuoles vary in size and number. Single vacuoles may attain a large size, as much as 20 to 25μ, and may lead to extreme distention of the nucleus. There are generally several vacuoles, and there may be as many as 6 or 8 in a single nucleus. Occasionally a cell has been met with in which the nucleus was ballooned out by a single large vacuole, filled with numerous droplets—an actual "physaliphorous nucleus." Nuclear vacuolation is most frequent in the more cellular parts, and it is absent where mucoid degeneration is advanced.

The stroma is composed of fibrous tissue, which in places shows advanced hyaline degeneration. Here and there are areas of extensive lymphocyte and plasma cell infiltration, with varying numbers of polymorphs, eosinophils, and mast cells. Blood vessels are fairly numerous, and small recent hæmorrhages are frequent. Some of the areas of hæmorrhage are of larger size, the blood having broken through into the interior of the alveoli. Former hæmorrhages are indicated by collections of endothelial cells filled with yellowish-brown pigment.

The hyaline change is best seen in those portions of the tumor in which mucoid degeneration of the parenchyma is advanced.

At the periphery, the tumor is enclosed by a dense fibrous capsule of varying thickness. Elastic fibers are present only in the outer layers of the capsule, not in the walls of the alveoli, or elsewhere throughout the tumor.

In Stewart's case, there was no evidence of invasion of the vessels by the growth, as has been described by some observers.

The following are Professor Stewart's general conclusions, and, except that a wider observation has shown that similar tumors may occur in sites additional to those specified by him in his original paper, it may be said that these conclusions are now pretty generally accepted. We quote them here in full:

1. "Chordoma is a tumor arising from relics of the notochord, and is met with chiefly in the neighborhood of the spheno-occipital synchondrosis and in the sacrococcygeal region.

2. "Both simple and malignant forms occur, the latter being much the more common. Even the malignant varieties are usually of slow growth and long continued course, especially those occurring in the sacro-coccygeal region. They tend to recur after removal, and cause death chiefly by their local effect, dissemination being quite exceptional.

3. "Intracranial clivus tumors, by virtue of their position, are much more serious than sacro-coccygeal, their average duration, from the first onset of symptoms, being about two years, as compared with nine years in the latter group.[1]

4. "The histological characters are distinctive. The tumor is alveolar in structure, and the parenchyma, usually of epithelial type, is composed of cells which become the seat of mucoid degeneration at a very early stage of their development. The mucoid change ultimately progresses to an extreme degree, and is comparable to that seen in the nucleus pulposus of an intervertebral disc. In malignant cases, the nuclei show great variation in size and in depth of staining, and nuclear vacuolation may be present."

THE NOTOCHORDAL ORIGIN OF CHORDOMATA

The developmental relation between these tumors and the notochord has gained, in recent years, an increasing degree of support, both on embryological grounds and by reason of the remarkable resemblances between the histological characters of the tumors and those of the notochord, or of such chordal rests as exist in the nuclei pulposi.

In his most recent paper on "Chordoma of the Vertebral Column," Cappell has an interesting note on the development of the notochord in certain of the lowest vertebrate forms, and on some aberrations of this development which have been observed by different workers. He describes also, and illustrates, the resemblances between the histological features in one of his cervical cases and the appearances characteristic of the developing notochord in Lepidosiren.

He points out that the notochord, at an early stage of its development, consists of a solid rod of epithelial cells extending from infundibulum to cauda in the embryo. In the cytoplasm, fluid vacuoles accumulate until the cell body becomes turgescent, this fluid inflation of the cell elements of the primitive skeleton being responsible for supplying the firmness necessary for its function of support.

He describes how the notochord is surrounded by a double sheath, the outer portion, which he terms the *primary sheath*, consisting of flattened cells of as yet undetermined origin (doubtfully notochordal or mesoblastic) and within this a *secondary sheath* formed by a mucoid material, which he believes to be secreted from the more superficially placed cells of the notochord. This secondary sheath forms a layer of fairly uniform thickness around the central notochordal cells. The more central cells retain their secretion within their cytoplasm, and become, in consequence, distended enormously by globules of doubtful nature.

Cappell has found that these appearances in the different stages of the ontogeny of the notochord are reproduced with striking fidelity in the histological characters of one of his cervical cases. To quote from his paper:

"There are" (in some places) "solid areas of clearly demarcated epithelial cells such as are found in the notochord in the second stage of its development. Later, the cells begin to differentiate, the characteristic mucinous secretion of notochordal cells appears, and actual physaliphorous cells are formed. In other places, the secretion is poured out freely into the intercellular spaces, the cells become shrunken, and the appearance of the notochord at a more advanced stage of development is reproduced

[1] This obviously refers to the "Malignant Spheno-Occipital Chordoma," and not to the "Ecchordosis Physaliphora Spheno-Occipitalis."

in an exaggerated degree. Lastly, just as when the notochord becomes enclosed in the centers of the intervertebral discs, to form the nuclei pulposi, the cells of the tumor become modified to form irregular syncytial strands with many large vacuoles of unknown nature."

As Cappell says:

"The presence of very definite sheaths round the smallest invasive elements of the tumor is a striking example of the reversion of tumor cells to a stage far back, not only in the ontogeny of the individual, but also in the phylogeny of the vertebrates."

Cappell figures a number of these appearances in a series of photomicrographs, and a critical examination of his illustrations constrains one to admit that they go far to support the view that the histological resemblances of the minute histology of the particular tumor under consideration to the histology of the developing notochord furnish strong presumptive evidence of a developmental relation between the two.

Cappell has a further interesting note regarding the manner in which chordal tissue may develop into tumor formation. He is clear that in his two cervical cases the tumor has originated *in* the affected vertebral body, or *on* its anterior or posterior aspect, rather than in notochordal cells persisting in the intervertebral discs. In this connection, he recalls the fact that the anterior end of the notochord in man, instead of being uniformly enclosed within the basal skull cartilage, is in part infrabasal, so that it lies immediately above the epithelial roof of the pharynx, reentering the basal skull cartilage in front of this point. This infrabasal part disappears earlier than the other portions of the notochord and is present usually only between the 12 millimeter and the 18 millimeter stages of the embryo. That such a relationship of notochord to basal cartilages and pharynx is recognized as normal is borne out by the fact, as Cappell points out, that it is figured in standard works on embryology. He believes that the establishment of such connections affords ground for suggesting a possible origin for cases such as his first cervical one.

Developmental abnormalities at lower levels have been recorded by different writers. Cappell cites some of these observations, such as those of Peyron, Dunet, Linck, and Warstadt, showing the existence of chordal rests beneath the perichondrium of the sacral bones; on the anterior or posterior surfaces of these—in the mesenchyme; as small protrusions in the lumbar bodies of human fetuses; as fine connecting strands between the successive nuclei pulposi, passing through the intervening vertebral bodies; and even in the form of small branches from these connecting strands, passing toward the anterior or posterior aspects of the vertebral bodies.

Finally, Cappell has found, in the lumbar region of a 21 millimeter embryo, small strands of notochordal cells issuing from the central core connecting the notochordal masses in the intervertebral discs, and passing ventrally, laterally, and even dorsally. He has not been able, however, to demonstrate that they led to any foci of chordal tissue on the exterior of the vertebræ. He figures a section of a lumbar vertebra, showing a protrusion of the sheath and cells of the central notochord, passing laterally to become lost gradually among the cells of the developing cartilage, and he suggests that such observations support the view that there exists normally, in this central thread of chordal tissue, perforating or channeling all the vertebral bodies, a sufficient basis from which notochordal tumors in the vertebral bodies may arise.

VENOUS DILATATIONS AND OTHER INTRASPINAL VESSEL ALTERATIONS, INCLUDING TRUE ANGIOMATA, WITH SIGNS AND SYMPTOMS OF CORD COMPRESSION

A Report of Four Cases With a Review of the Literature[1]

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THERE is a small group of alterations and malformations of spinal cord vessels which give rise to signs and symptoms of spinal cord compression. This group includes the localized dilatations or aneurismal formations of spinal cord vessels and the true hæmangiomata, which are with rare exception recognized only on the operating table or at postmortem examination. They are more often diagnosed as spinal cord tumors, since their clinical manifestations, by virtue of their focalizing character, are not unlike those found in spinal cord compression. Before the advent of manometric tracings and intraspinal lipiodolography, such errors in diagnosis were obviously unavoidable, since there were as yet no reliable diagnostic features for the correct identification of the vascular origin of such tumor-like structures, and since they provoked symptoms such as noted in various forms of intraspinal neoplasm.

It was in search of some helpful pathognomonic signs in such vascular lesions that this study was undertaken. The study was concerned mainly with the focal dilatations of spinal cord veins and included an analysis of the clinical manifestations and anatomical findings in a large series of cases collected from the literature and an account of our own cases. However, while our own material as well as the majority of that recorded in the literature belongs to the group in which the vessel changes are in the nature of venous dilatations, we nevertheless have extended this survey to include all examples of tumor-like, vascular alteration, neoplastic or non-neoplastic in character. Thus we have added to the rather large assembly of various types of allied conditions, such as arterial and arteriovenous aneurisms which differ from the former group mainly in their anatomical features and the true hæmangiomata with which the former two groups are often confused. These will be discussed separately and for convenience of description under the following headings: Group I, venous dilatations; Group II, arterial or arteriovenous aneurisms, Group III, hæmangioma—(a) intramedullary, (b) extramedullary (pial), (c) extradural, and (d) vertebral.

GROUP I—DILATATIONS OF SPINAL VEINS

This is the largest group assembled. The individual members present anatomical variations which are responsible for a corresponding assortment of terms under which they are described. Thus we have a series of names, such as: pial hæmorrhoids (Gaupp), cirsoid aneurisms of the spinal veins (Raymond and Cestan), varicose dilatations of spinal veins (Jumenti and Valensi), angioma venosum racemosum (Krause), and angiomata, all of which, however, can justifiably be grouped together under the single term of dilatations (varicose) of the spinal veins.

The occurrence of this form of spinal vessel alteration is considered rare by almost every one who has written on this subject. However, no such conclusion can be drawn from the meager material available in the literature. The scarcity of material is in great measure due to a lack of opportunity for postmortem examination of well studied neurologic material and, above all, to the indifference of the pathologist to the examination of the intraspinal contents in non-neurologic cases. To obtain some conception as to the frequency with which dilatations, tortuosities, and other anomalies of pial veins of the spinal cord occur, we must turn mainly to the work

[1] From the neurological service and laboratories of Mount Sinai Hospital, New York.

of Kadyi. In a general study of the vascular supply in a series of 26 cases of human spinal cords, he found that dilatations and tortuosities of larger or smaller veins of the spinal cord are not uncommon. In 8 patients various degrees and several types of varices of the spinal cord veins were noted. Even in spinal cords in which the veins presented a normal appearance he frequently noticed that the course of the veins on the dorsal aspect of the cord was somewhat tortuous. He, therefore, concluded that a careful study of a larger series of cases would disclose various transitional forms with gradual change from the normal condition to the most marked grade of varicosities in which the venous trunks and their branches form a most bizarre network and are often so extensive as to cover the surfaces of the spinal cord completely. He also described changes in the pia arachnoid membranes directly over the varices. The membranes were thickened and had the appearance of an old inflammatory lesion. This led him to assume that, at least in some instances, an inflammatory process might be responsible for the development of varicosities of the spinal veins. Unfortunately, he was unable to give any clinical data in the individual cases studied and could throw no light on the character of his material beyond the fact that the varicosities of the veins of the spinal cord were found only in cases in the far-advanced years of life. He also suggested the possibility that excessive muscular activity, particularly of the spinal muscles, caused an obstruction to the free circulation in the spinal canal, and was an important contributing factor in the production of varices on the surface of the spinal cord.

Kadyi described two types of varices. In one series of cases he found, on the dorsal surface of the cord, large tortuous veins which resembled in their arrangement loops of small intestines. The vessels covered the spinal cord in several layers, the smaller branches showing very little of this tortuosity and varicosity. Such a picture, in his opinion, could be explained only by an interference with the free return of venous blood from the spinal canal, the obstruction being very likely caused by excessive contraction of the muscles of the spinal column. In another group, the smaller vessels showed a high grade of tortuosity and formed veritable pools while the larger venous trunks were not enlarged and appeared narrowed in contrast with the dilated smaller branches. In such instances he found a distinct interruption between the ventral and dorsal venous anastomoses.

It occurred to him that the narrowing (and possibly complete obliteration) of the larger trunks caused the formation of a collateral circulation in the smaller venous channels with the result that the latter, because of the new burden, became widened, elongated, and tortuous. In some instances he noted that root veins, as large as the main trunks, became in some part of their course, very narrow or completely obliterated. In these cases he believed that the dilatation of the smaller branches was compensatory.

In a more recent analysis of the various views held as to the causative factors in the formations of phlebectasias, varicosities, and so-called venous angiomata, Benda does not give definite conclusions. He quotes Rokitansky, who stresses the mechanical hindrance to the blood flow as the all-important factor. Rokitansky enumerated a number of anatomical alterations which may bring about such an interference in circulation. Among them are: pressure upon a venous trunk by a tumor, obliteration of venous channels, unusual posture of the body causing a slowing down of the return blood flow, oft-repeated attacks of hyperæmias or inflammations in an organ, and finally inflammation of the veins themselves. But he also recognized that the purely mechanical factors did not explain all the phenomena observed in such venous dilatations. Some concomitant or pre-existing alteration in the involved veins must be considered as an important additional factor. Degenerative, sclerotic, or inflammatory changes are considered by many as the probable predisposing condition which, when acted upon by a local or general circulatory disturbance, determines the focal and somewhat circumscribed venous dilatations. The possibility of a pre-existing, congenital weakness in the vessel wall, as a contributary factor is dismissed by Benda as a conception unwarranted by known facts. Thus the present opinion does not vary greatly

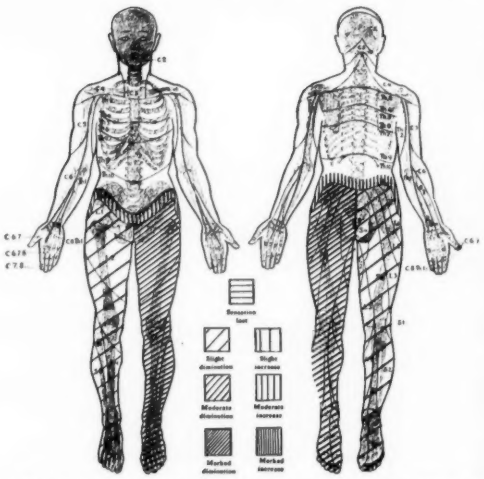

Fig. 1. Sensory chart indicating changes in pain and temperature sense. Case 1.

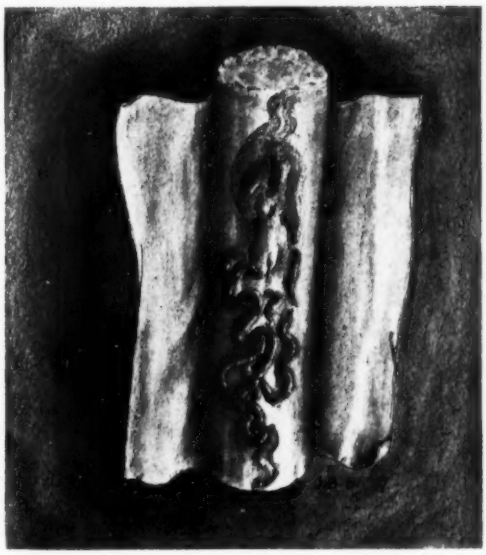

Fig. 2. Drawing showing the appearance of the dorsal surface of the cord. Case 1.

from the older views expressed by Kadyi, but it does coincide with the view held by Rokitansky that by impairing normal return of venous blood abnormal postures have an important bearing on venous dilatations.

Benda makes another valuable contribution to the subject of venous dilatation by offering a simple classification of such anomalous conditions. His classification brings order into the somewhat confusing terminology, and above all makes the interrelationship between the several varieties more obvious. He reduces the large assortment of venous anomalous dilatations to three forms:

2. *The phlebectasias*, which he describes as a form of diffuse widening of the lumen of the veins, in which the shape of the dilated veins depends upon the type of vessels involved. The widening of a large trunk will result in the cylindrical variety while widening of small branches gives rise to the so-called cirsoid or plexiform variety.

2. *The varicosities*, a form in which diffuse but irregular dilatations are characterized by circumscribed, ampullar, or sac-like formations. Usually these are not associated with the first form of venous alteration and may be regarded as a more advanced stage.

3. *The venous angiomata*. This is a type which is essentially a circumscribed conglomeration of dilated veins, with individual alterations which may fall into either one of the two previous groups, phlebectasias and varicosities. The angiomata differ, however, from the two other forms by inducing secondary changes, such as erosion or production of tissue alterations in the neighboring structures, and so acquire the character of tumor formation. Between these three forms, however, there are no distinct lines of demarcation, they are essentially transition stages of a similar process. A complete or partial closure of a large venous trunk, from whatever cause, leads to stasis in the blood column with the formation of a collateral circulation, and a resulting venous dilatation. In the larger vessels which are adjacent to the obstructed venous stem, the dilatation assumes a cylindrical form, while dilatation of smaller vessels results in the plexiform or cirsoid formation. It is the latter form which is most commonly seen in the pial vessels of the spinal cord. Because of their localized character, and because of their mechanical effect upon the spinal cord, they may easily fall into the group of the so-called "venous angiomata." It is quite obvious, however, that they are

Fig. 3. Section of the cord showing a number of tortuous and dilated pial veins. Photomicrograph ×12, hematoxylin. Case 1.

nothing more than circumscribed aggregations of dilated varicose veins and that they are not neoplastic in origin.

PERSONAL CASES

Case 1.—A. C., male, aged 59 years, was well until 7 months prior to his admission to the hospital (April 6, 1926), when one morning he awoke to find himself unable to move his legs. He improved at first but soon there was a progressive loss of power in his lower extremities so that he could no longer walk without support. Four months after the onset, in addition to further loss of power in the extremities, there developed difficulty in urination, loss of sexual power and a feeling of numbness of the dorsal surfaces of his feet.

The patient is unable to walk without aid. Both lower extremities are markedly paretic, the right more than the left, with a right foot drop. The musculature in the affected limbs is flabby and atrophied. The right knee jerks, both ankles jerk and the cremasteric reflexes cannot be elicited. The abdominal reflexes are active. There is a belt of hyperalgesia extending from the twelfth dorsal to the second lumbar (Fig. 1.) Below this level there is marked hypalgesia and thermohypæsthesia, which is more pronounced on the left side. Vibratory sense is impaired below the anterior superior spinous processes. There is percussion tenderness over the tenth dorsal. The electrical tests give normal responses. The manometric tests reveal no block, with an initial pressure of 290 millimeters, a slight rise on coughing (310 millimeters), a greater rise on straining (460 millimeters) and still higher on jugular compression (500 millimeters). The rises are prompt and are followed by an equally prompt fall in pressure. Serologic and other tests of blood and spinal fluid are negative. The X-ray examination of the spine shows no striking changes. The blood pressure in the right arm is 160-90 and in the left 135-75.

While under observation there was no material change in his condition. On May 7, 1926, he was discharged with the provisional diagnosis of extramedullary compression, probably due to a neoplasm on the right anterolateral aspect of the cord at the level of the twelfth dorsal. He was re-admitted on September 30, 1926, complaining of increasing difficulty in urination (hesitancy and interruption of the stream) marked constipation, œdema of his left leg, and frequent twitching in both lower extremities.

Re-examination at this time showed, that, added to previous findings, the left knee jerk and the lower abdominal reflexes were absent, with a loss of touch sense below the first lumbar. The electrical tests of the muscles of the lower limbs revealed a diminution to faradic and an increase to galvanic stimulation. The blood pressure in the right arm was 119-76 and in the left 84-56. The monometric tests revealed an initial pressure of 100 millimeters with normal rises and falls on coughing, straining, and on jugular compression. There was no xanthochromia. The urine showed a faint trace of albumin and a few white blood corpuscles, 73 per cent polynuclears, 20 per cent lymphocytes, and 7 per cent monocytes. At this time a degenerative process in the spinal cord was considered as the more probable character

Fig. 4. Section of the cord on level of the aneurisma venous dilatations, showing numerous dilated venous channels. Photomicrograph ×65, hematoxylin. Case 1.

of the lesion, though a neoplasm was not entirely excluded.

The patient's incontinence was soon followed by signs of cystitis, and pus was found in the urine. His temperature rose to 103 degrees for about 10 days, gradually returning to normal. On irrigation of the patient's bladder a fragment of thick nucoid material was recovered, which the pathologist reported contained carcinomatous (?) tissue. Immediately the possibility of malignancy with metastasis to the spine was suspected, although no primary focus could be found. On November 1, the patient suddenly had a convulsive seizure, lost consciousness, and appeared to have a right central facial paresis. The fundi showed angiosclerosis and a hæmorrhage in the region of the left macula. The patient's condition declined rapidly. He passed into deep coma and died on the fifth day after the cerebral accident, without regaining consciousness.

Anatomical findings. Only a small portion of the cord could be removed. It included the lower lumbar and the adjacent sacral segments. On opening the dural sac, there was found on the dorsal surface of the cord, a fairly circumscribed but very prominent mass of convoluted, tortuous pial veins. They were intimately adherent to and apparently invaded the structure of the cord (Fig. 2). A histological study revealed alterations in the pial veins as well as in the vessels and the substance of the spinal cord. The dorsal pial veins showed the most prominent alterations. They were markedly dilated and highly irregular in outline (Fig. 3). This, of course, corresponds to the tortuosity of the vessels already noted on gross inspection. Some of the veins on the surface of the cord had an oval

Fig. 5. Section of the cord showing disintegration of its normal cell structure and the increase in glial contents. Photomicrograph ×225, silver carbonate stain. Case 1.

Fig. 6. Section of the cord showing hyalinized vessels. Photomicrograph ×55, hematoxylin-eosin. Case 1.

or round form, others were flattened or crescentic in outline, while still others were sacculated or so malformed as to engulf a neighboring vessel. In addition to this gross variability in size and shape, the vessels showed also striking changes in their coats. The media was especially involved; it was hypertrophied and consisted of many cross and oblique connective tissue fibers, among which there were seen many smooth muscle fibers. The latter showed many focal quantitative variations, often approaching an abundance seen in arteries. The elastic fibers were also quite numerous. The intima also showed mild alterations in the nature of diffused hyalinization. The endothelial lining, however, was intact, consisting most commonly of a single layer of cells, but, occasionally, where the vessel outline was deformed, it gave the impression of pseudo-stratified structure. The adventitia showed no marked changes; there was no mesodermal reaction to suggest an inflammatory lesion.

The pial arteries showed no changes aside from a mild hyalinization of the intima.

The leptomeninges were distinctly thickened. There was slight fibrosis and infiltration with macrophages, fibroblasts, and an occasional lymphocyte.

The substance of the cord showed extensive disorganization in both the white and gray matter. This was due to the marked loss of parenchyma and also to an invasion by numerous distended vessel channels (Fig. 4). Of the gray matter there are left only a few islands of nerve tissue with only a few nerve-cells retaining their normal outline and structure. The majority of the residual cells showed disintegration, sclerosis, and some of them even

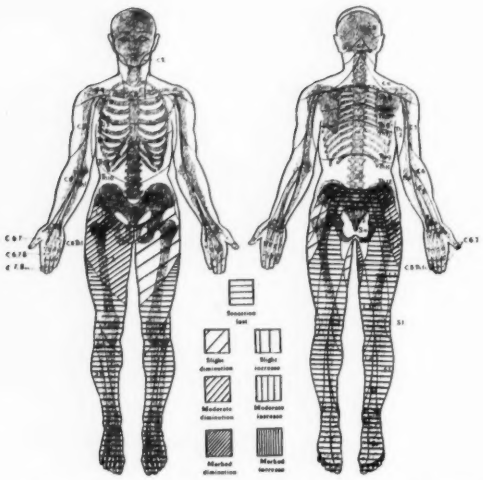

Fig. 7. Sensory chart showing changes in pain and temperature sense. Case 2.

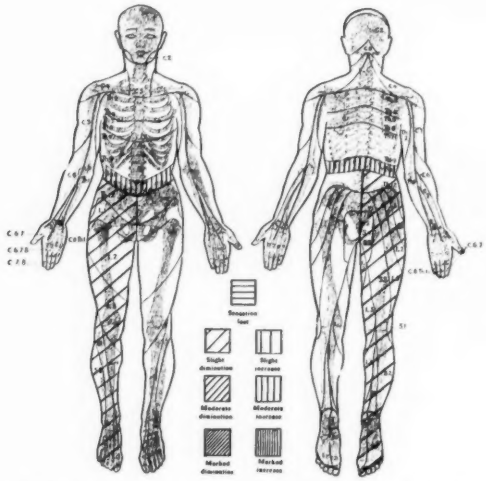

Fig. 10. Sensory chart illustrating changes in pain and temperature sense. Case 4.

calcification, and were surrounded by zones of moderate glial hyperplasia in which astrocytes formed the dominating element (Fig. 5). The white matter was the seat of a moderate gliosis and only a few islands of partially preserved myelin fibers remained.

Because of the loss of parenchyma, the vessels in the substance of the spinal cord stood out very prominently. There were many distended and deformed venous channels and distended and otherwise altered capillaries and small-sized arteries were quite numerous. Some of the veins, particularly at the periphery of the cord, showed open communication or fusion with the pial vessels on the surface of the cord. Similarly groups of intercommunicating or fused blood channels with hyalinization in their

Fig. 8. Dilated veins found in Case 2.

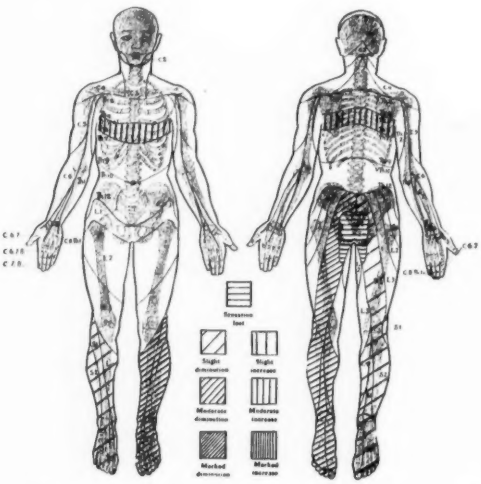

Fig. 9. Changes in pain and temperature sense as disclosed in Case 3.

Fig. 11. Dilated veins found in Case 4.

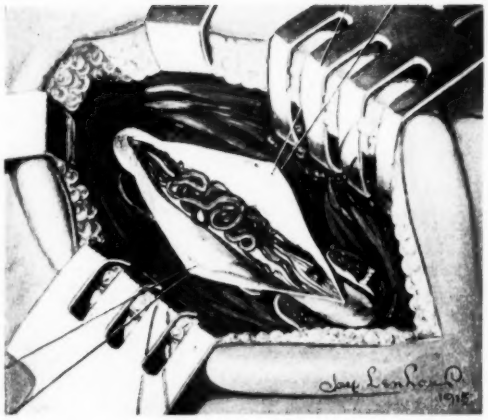

Fig. 12. Drawing illustrating an arteriovenous aneurism of the spinal cord. (Elsberg's case.)

walls were seen within the substance of the cord. They were frequently surrounded by zones of fibrosis. The walls of the arteries showed still more advanced degenerative changes (Fig. 6). There were frequently groups of four to six vessels surrounded by an area of fibrosis, having the appearance of a vascular island.

The anatomical findings briefly restated were: (1) marked varicose dilatation of the dorsal pial veins, (2) extension of this venous dilatation into the vessels of the spinal cord, (3) degenerative changes in the arteries and veins of the nature of moderate arteriosclerosis and phlebosclerosis, (4) marked disorganization and disintegration of the spinal cord substance.

Several clinical features assume greater significance after the pathological process is clear. The rather sudden onset, the progressive course and the terminal cerebral manifestations associated with convulsive seizures are evidence of a generalized vascular disease which affected also the spinal vessels.

CASE 2. M. B., aged 50 years, except for an attack of influenza 5 years previously, was well until May, 1923. At that time, while in bed, he suddenly experienced a sensation of coldness and of "pins and needles" in his toes. These sensory disturbances continued and were soon followed by gradual loss of power in the right leg. Shortly thereafter, he developed obstinate constipation and somewhat later fæcal incontinence. The condition remained unchanged for about 2 years, when, following an appendectomy for a ruptured appendix, he lost control of the bladder and developed painful and recurring cramp-like attacks in his right leg. The numbness in that leg became very marked so that

Fig. 13. Drawing showing location and type of hæmangioma. Case 6, Table IV.

he would frequently burn himself because of failure to recognize the higher degrees of heat. Three and a half years after the onset of symptoms he entered the Mount Sinai Hospital.

There is motor weakness in both legs, greater on the right. There is a right foot drop. The deep reflexes in the lower extremities are barely elicited even with reinforcement; no Babinski; and lower abdominal reflexes absent. There is hypalgesia below the first lumbar with partial analgesia on the right side. The disturbance in temperature sense follows the same distribution; vibratory sense is lost below the anterior superior spines; postural sense is lost in the toes. The manometric test shows no subarachnoid block with an initial pressure of 180 millimeters; pressure on coughing, 190 millimeters, on straining 200 millimeters, on jugular compression 220 millimeters. The cerebrospinal fluid is cloudy; it contains six cells per cubic millimeter. Wassermann test of blood and cerebrospinal fluid is negative. Lipiodol injection into the cisterna magna showed no block.

The patient's stay in the hospital was marked by loss of his right knee jerk, the appearance of a bilateral Babinski sign, hyperactivity of the ankle jerks and the appearance now of hypalgesia from the first to the third lumbar, greater on the right, bilateral analgesia below the second lumbar with normal sensation retained in the third to fifth sacral (Fig. 7). The legs became spastic. Electric tests gave normal responses. The picture was considered as that of an extramedullary neoplasm, affecting mainly the roots of the cauda equina.

An exploratory laminectomy was performed by Dr. Elsberg. When the dura was opened and traction put on it, there was active bleeding from the dura, and a mass of enormously dilated veins covering the upper two-thirds of the exposed area appeared (Fig. 8). In the lower part of the wound a little of the cord, yellowish in color, could be seen as well as numerous small vessels entering and

Fig. 14.

Fig. 14. Gross and microscopic appearance of hæmangioma. Case 6, Table IV.

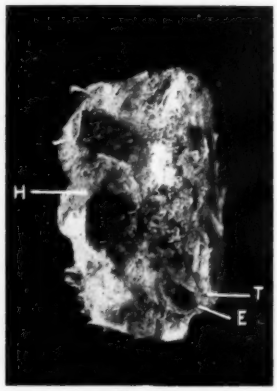

Fig. 15.

Fig. 15. Vertebral hæmangioma, *H*, associated with a similar epidural tumor, *E.I.* Case 7, Table V.

leaving the cord. In the middle of the exposed area there was one prominent loop of veins which projected backward across the cord for at least 1 centimeter. Probing forward and upward disclosed no obstruction. The arch of another vertebra was then removed and the incision of the dura extended upward, thus exposing further masses of enlarged veins covering the surface of the cord so that the cord could not be seen; further probing upward failed to show any obstruction. The wound was then sutured.

In the further history of the case, Dr. Elsberg states: "A typical extensive varicosity of the veins of the cord, which the writer described a number of years ago under the heading of varicose veins of the cord. All the vessels were thin walled, many of them entering and leaving the cord substance so that it was inadvisable to attempt to ligate any of the vessels of the cord, as the effect on the cord of shutting off its blood supply from even these abnormal vessels would be certain to cause a marked exaggeration of the symptoms and possibly a transverse myelitis."

Following the operation, the patient received radium therapy and slowly regained some power in his legs. At the time of his discharge on January 20, 1927, he was able to get about fairly well, but still there was unquestionable weakness in the lower extremities. He showed diminished deep reflexes in the right leg, absent lower abdominals, bilateral Babinski, and a sensory status as on admission. He was readmitted to another neurological service, where another lipiodol injection was done, but no block was found. His condition declined progressively, and he developed decubitus ulcers, total incontinence, became septic, and died on November 1, 1927.

Of note in this case is the rather sudden onset of paræsthesias which were soon followed by motor disturbances. The bladder and rectal involvement coming on later, indicated an extension of the lesion into the depth of the cord. It is of significance that there was no subarachnoid block.

Case 3. J. S., male, aged 53 years, suddenly developed 2 years prior to his admission to the hospital (March 10, 1924), severe pain about the rectum, for the relief of which an hæmorrhoidectomy was performed. Soon after the operation, he began to experience pain in the ankles which shortly after spread to both lower extremities and was accompanied by paræsthesias in the soles of his feet and difficulty in micturition and weakness in the lower extremities. A neuro-surgeon made the diagnosis of a spinal cord tumor and performed a laminectomy (August, 1923) in the region of the third and fourth lumbar processes. He found no tumor, but instead, an increased vascularity of the cord, and adhesions of the roots of the cauda equina to the dura. The decompression was followed by a temporary relief, but in the early part of 1924 there returned pain of greater intensity associated with cramp-like attacks in his feet. To the difficulty in micturation, there was now added marked constipation.

The pupils are irregular, left larger than the right. Abdominal reflexes are markedly diminished on the right side. Both knee jerks are active and equal, both ankle jerks hyperactive. There is bilateral ankle clonus and bilateral Babinski. The lower extremities are weak, left more than right, they are spastic, and there is some atrophy of the muscles on the left side. Complete left and partial right foot drop is present. There is a narrow zone of hyperalgesia at the fourth to sixth dorsals, hypalgesia from the fourth lumbar down, more marked on the left side. There is complete analgesia in distribution of the second to fifth sacrals and a reduction in

the temperature sense over the same areas. Vibratory and postural sense is lost below the knees (Fig. 9). The spinal fluid shows increase in pressure, but no block, no xanthochromia, and but 2 cells per cubic millimeter The blood and spinal fluid Wassermann tests are negative.

The findings were regarded as suspicious of an extramedullary neoplastic lesion compressing the spinal cord. A laminectomy was performed by Dr. Elsberg, the spinal canal being opened at seventh to eighth dorsals. The dura was incised and the cord was exposed. A very large varicose vein was found lying along the left side of the dorsal surface of the cord. Cerebrospinal fluid escaped freely from above and probing upward proved negative. The spinous processes the ninth, tenth, eleventh, and twelfth dorsals were then removed and the same large vein was seen running down toward the cauda equina, and there were found some fine adhesions between the membranes and cord. No tumor was found and the postoperative diagnosis was: varicose vein of the cord presenting the picture of spinal cord tumor. The patient made an uneventful recovery, showed slight improvement, and his pains were considerably relieved.

Of significance was the sudden onset followed by a fairly long clinical course. Pain in the perianal region and in the extremities was an early symptom and was soon followed by bladder difficulty. The objective findings localized the level of lesion, pointed to its extramedullary situation, and had some features of a neoplasm. The operative findings, however, though not as striking as in the first case, differing mainly in degree rather than in the actual anatomical alterations are better understood in the light of Case 1, and satisfactorily explain the clinical manifestations. The absence of subarachnoid block or xanthochromia deserved more weight than was given to it at the time.

Case 4. H. G., male, aged 62 years, was in the hospital three times. On his first admission (August 10, 1922) he gave a history of weakness in his legs (greater on the left side) and 2 years' duration. This was followed one year later by pain and paræsthesias in the lower extremities (more marked on the right side) and more recently (4 months before admission) by difficulty in urination. Examination at this time showed a paraplegic gait with spasticity in the left lower extremity greater than in the right, diminished lower abdominal reflexes, absent cremasteric reflexes, hyperactive knee and ankle jerks, bilateral Babinski sign, left ankle clonus, impairment of pain and temperature sense on the right side below the twelfth dorsal and a vague lever of hyperalgesia at the twelfth dorsal (Fig. 10). There were varicose veins in both legs. The blood pressure was systolic 134, diastolic 74. The blood and cerebrospinal fluid Wassermann tests were negative, and the urine was normal. After a short stay, he left the hospital with the diagnosis of "spinal arteriosclerosis," or possible spinal cord tumor. He was re-admitted 3 months later (Feb. 20, 1923), when he showed increased weakness in his left leg and an increase in the intensity of the burning pain in his right leg. His bladder control on the other hand showed some improvement. The neurologic status at this time showed but little change from that of his previous admission except that there was now a definite loss of vibratory sense in the left leg below the knee and over the sacral vertebræ. X-ray examination of the spine disclosed mild spondylitis. At this time an intramedullary spinal cord tumor at the level of the twelfth dorsal was regarded as the most probable diagnosis. Operation was suggested but the patient went home to consider it. He returned to the hospital 4 months later (July 5, 1923), when upon examination, he showed slight change in his status. The sensory disturbances now extended to a slightly higher level (tenth dorsal), the knee jerks and anke jerks were hyperactive and more so on the left side, the lower abdominal reflexes were absent, there was a left ankle clonus, a left Babinski sign with an equivocal right Babinski sign and spasticity and weakness in both lower extremities which was more marked on the left side. The sensory level, however, was not very definite and because of that, the diagnosis of an "extensive degenerative disease of the cord with multiple foci" was favored.

Laminectomy was performed by Dr. Neuhof, who removed the spinous processes from the second to the fifth dorsal vertebræ and, on exposing the cord, found on the right side of the cord, opposite the third and fourth dorsals, a contorted mass of dilated veins (Fig. 11). At its lower border this mass merged into veins, while at its upper end, there was a series of smaller veins which were closely attached to the posterior surface of the cord, on one hand, and to the aneurismal mass, on the other. The anterior limits of the mass could not be determined but it was evident that it extended well around to the anterior surface of the cord. The latter was displaced by the aneurismal mass to the left. Ligation of the vessels was felt unjustifiable, for it was feared that such a procedure would cause considerable damage to the cord. Exploration above and below the vascular tumor proved negative. The patient made an uneventful recovery from the operation but showed no change in the neurologic status. Two months later he was tranferred to another institution for deep radiotherapy.

The outstanding clinical features in this case are the protracted clinical course of 3 years' duration with gradual unfolding of the manifestations of cord compression, and the

terminal appearance of signs of intramedullary involvement. Of significance also are the negative cerebrospinal fluid findings, including the lack of xanthochromia. The operative findings place the case clearly with instances of varicosities of spinal vessels.

CASES COLLECTED FROM THE LITERATURE

The clinical records and the anatomical findings in 24 cases collected from the literature are incorporated in Table I. It is believed that all available instances of venous dilatations with compression or direct invasion of the spinal cord, aside from those, which are not accompanied by clinical records, have been included in this table. In it are recorded the more striking clinical features and only brief references to the anatomical alterations. For more detailed information the reader is referred to the original articles.

SUMMARY AND GENERAL COMMENT ON GROUP I

An analysis of the clinical features, presented by the material in the first group, reveals very few data of diagnostic value. However, some suggestive leads are obtained from a consideration of some clinical data under the following headings:

Age. In the group of 28 cases the ages of the patients are distributed as follows:

	Cases
In second decade (the earliest age incidence)	2
In third decade	6
In fourth decade	8
In fifth decade	3
In sixth decade	6
In seventh decade	1

(In two cases the age was not given.)

The largest number of cases occurred in the third, fourth, and sixth decades, the smallest number in the second and seventh decades. Thus it appears that the lesion is most frequent during the more active years of adult life, between the ages of 25 and 50 years.

Sex. It is significant that of the group of 28 cases 21 occurred in males. This again, would also strongly favor the belief that the physically more active are more likely to develop this form of lesion.

Trauma. In only 4 cases was trauma recorded and in only 2 instances did it directly precede the onset of symptoms. This would minimize the importance of trauma as a causative factor.

Onset. In 15 cases, the onset was acute, almost precipitate, while in 9 it was less abrupt, perhaps subacute in character; in 4 cases the signs and symptoms developed gradually and insidiously.

Initial signs. Pain, motor weakness, and paræsthesias stand out most prominently as initial signs, with pain as the most frequent early symptom. It had occurred as the first manifestation in 13 cases, while motor weakness occurred in 12 cases, and paræsthesias only in 3 cases. Each of these signs and symptoms may have occurred alone or in association with one or more of the other manifestations, as for example where motor weakness was the more prominent symptom, pain or paræsthesias may also have been present.

Type of paralysis. The clinical histories of the cases collected from the literature are not particularly clear on this phase of the neurologic picture. It is certain that of 23 cases only 11 cases showed the flaccid paralysis type, while 7 showed spastic paralysis. The paralysis involved mainly the lower extremities. In only one case was there also weakness in one arm. Footdrop was a common occurrence.

Sensory changes. Here also the observations are not very accurate and yield few instructive findings. Sensory disturbances, however, in the great majority of instances were found to correspond to the level at which the lesion was found.

Course. The course is always progressive. It is not an uncommon feature for the clinical picture to gain momentum in its evolution at the very beginning of the illness then come to a stationary period of variable length, after which further but slow progression takes place. The clinical course may extend over variable lengths of time. It may be short and of but a few months' duration, or so long as to spread over a period of 24 years. The height of the clinical picture may occur at any period in the clinical course.

The more common levels of lesions were: lumbar cord, 7 cases; lower dorsal cord, 11 cases; mid-dorsal cord, 5 cases; upper dorsal cord, 1 case; cervico-dorsal cord, 2 cases. It would

seem that the most common levels are at the lower dorsal and the lumbar segments.

Co-existence of nævi. In only one instance was a nævus found which helped in the recognition of the character of the disease.

Laboratory findings. Serologic tests, cytologic studies, manometric estimations of the spinal fluid and lipiodol tests have not been carried out systematically and hence offer no data which would permit of an analysis. In a few cases in which such findings were reported, they were uniformly negative.

It is obvious that none of the above data may be used as diagnostic criteria, but may be taken in consideration in atypical instances of cord compression along with other diagnostic possibilities. It is, however, significant that in a large number of instances, laminectomy which was carried out in the belief that a spinal cord tumor would be found, had given satisfactory results in 9 of the 28 cases, with partial or complete recovery. A decompression alone or decompression associated with very judicious and conservative removal of veins was responsible for the improvement. In only 3 cases was there no improvement following decompression by laminectomy. In 8 cases laminectomy was followed by a fatal issue. Here the responsibility may be traced to surgical procedure which was somewhat too aggressive, the radical measures having included resection and removal of the venous dilatations. Removal of such veins is always fraught with danger since in many instances these vessels invade the spinal cord, and interference with such vessels will often lead to vascular disturbances and degenerative changes in the cord itself.

GROUP II. ARTERIAL OR ARTERIOVENOUS ANEURISMS OF SPINAL VESSELS

This is a smaller group and its members differ little clinically from those in the preceding one. Their anatomical features, the limitation of the alteration to a circumscribed area of a single vessel, however, justify their grouping under the separate heading. Little need be said here about the causative or predisposing factors responsible for such vessel alteration of spinal arteries, for they are not likely to differ from those causing similar changes in vessels at the base of the brain or in vessels elsewhere in the organism. Hence we pass on to the description of the individual cases.

Brasch's (25) patient was well up to the age of 59 years, when he rapidly developed weakness in his legs and somewhat later, incontinence of urine with pain in the gluteal and perineal regions which radiated down the legs. At the end of 2 years the patient was no longer able to walk. A neurologic examination at this time showed a mild, left central, weakness of the face, tremor of the hands, paralysis of both lower extremities (with but slight movement in the toes still retained) absent knee-jerks, anæsthesia in the lower extremities, and hypæsthesia in the lower abdomen. The patient declined rapidly and died 2 days after admission to the hospital.

Anatomical findings. The dura was defective from the fifth dorsal vertebra down the posterior surface of the cord thus permitting the penetration of large thickened tortuous pial vessels. From this point on, the vessels were traced downward along the dorsal surface into a mass made up of numerous vascular coils and loops. At the first lumbar segment they were seen to become continuous with a thickened tortuous artery, which soon divided into two smaller branches. On the ventral surface of the cord there was found a less markedly tortuous vessel, winding its way from the second to the first dorsal where it penetrated the dura, reached the pia, and then continued down to the third where it divided into two small branches. The vessels had markedly thickened muscle coats and widened lumina, but the intima was normal and the adventitia only slightly thickened. Numerous vessel loops penetrated the depth of the posterior surface of the cord, with the coils of vessels taking the form of kidney glomeruli. The smallest vessels showed marked thickening of the muscle coat which frequently caused occlusion of the vessel lumen. In some of the pial vessels calcification of the muscle layer was noted.

Though the case is described as an instance in which the alterations occurred in the arterial tree, the clinical manifestations as already pointed out differ little from those seen in venous dilatation of the cord vessels.

Guizzetti and Cordero's case (26) is a very unusual instance of "hæmatomyelia" as the result of the bursting of an aneurism of the ventral spinal artery. The sac of the aneurism rested between the first and second dorsal vertebræ and was nearly 2 centimeters long and was oval in shape. There was hæmorrhagic extravasation as far up as the fifth cervical and spread down to the ninth dorsal. The wall of the aneurism consisted of hyaline connective tissues with hardly any trace of smooth muscle or elastic tissue. About the aneurismal sac was a conglomeration of very

TABLE I.—DILATATIONS OF SPINAL VEINS, WITH COMPRESSION OR OTHER INVOLVEMENT OF THE SPINAL CORD

Case No. Author	Age sex	Onset and early manifestations	Late manifestations and their duration	Neurologic findings and diagnosis	Course and termination	Anatomical findings
1 Gaupp	45 F	Injury at age of 7 years. At 21 sudden attack of dizziness and syncope. Two months later weakness in both legs. Temporary recovery	Progressive loss of power in the left leg over a period of 10 years; later power lost also in the right leg. Sensation of coldness in the left leg. Loss of control of sphincters (24 years)	Paralysis of the right leg and paresis of the left; anæsthesia of the right leg and hyperæsthesia of the left; vascomotor paralysis of both legs; contractures at the hips. Diagnosis cerebrospinal meningitis	Decubitus ulcers Sepsis Death	Varicose veins over lumbo sacral segments of the cord.
2 Raymond and Cestan	30 M	At the age of 17 sudden onset of sharp pain in back of the neck, radiating down the right arm; later weakness of the right arm and leg	Progressive weakness in the left arm and leg paræsthesias in the left arm (13 years)	Weakness in both legs; atrophy of the right arm. Radicular pain in distribution of the right cervical and brachial plexus. Diagnosis: myelitis and later spinal cord compression	Laminectomy was planned but patient succumbed to acute pulmonary infection Death	Varicose veins compressing the cervical cord and penetrating its substance. Alterations also in arteries.
3 Jumenti and Valensi	40 F	Sudden development of flaccid paraplegia in the lower extremities	Flaccid paraplegia persisted (Very short duration)	Flaccid paralysis of both lower extremities; loss of deep reflexes in the lower extremities	Death (3 years after onset)	Tortuous mass of varicose veins at C-5 to D-7.
4 Lindemann	59 F	Gradual onset of weakness in the legs	Complete loss of power in the legs; loss of sphincteric control (1 year)	Flaccid paraplegia; loss of abdominal reflexes; loss of knee and ankle jerks; atrophy of legs; sensory loss below umbilicus. Diagnosis: transverse lesion of the cord	Decubitus ulcers Death	Varicose vein compressing lumbar and dorsal segment of the cord.
5 Krause	34 M	Weakness of the lower extremities	Complete paraplegia (7 years)	Paralysis of both lower extremities. Diagnosis: tumor of the spinal cord	Laminectomy and partial removal of mass of veins Death	At operative field, the cord was reduced to thin shred; above and below varicose veins.
6 Elsberg	23 M	Sudden onset of cramp-like pain on left side of the lower abdomen radiating to left leg	Increasing loss of power and spasticity of both legs (3 months)	Both legs weak, left more than right; absent abdominals. Knee jerks exaggerated, left more than right. Babinski on left side, exhaustible ankle clonus on left. Loss of pain and temp. below D-9. Diagnosis: focal lesion D-8-9	Laminectomy. Larger varicose veins removed Recovery	
7 Elsberg	27 F	Gradual onset of weakness in the legs	Flaccid paraplegia; impairment of bowel and bladder control (1¼ years)	Flaccid paraplegia; impaired sphincter control; sensory loss over S-3, S-4 and S-5; loss of knee and ankle jerks	Laminectomy and removal of varicose vein between cauda equina roots Recovery	Large varicose vein removed from between roots of cauda equina.
8 Meyer and Kohler	29 M	At the age of 7 a sudden attack of pain in the left shoulder, radiating down the left arm; pain increased in intensity	Weakness of the left arm set in followed by atrophy; later weakness and atrophy of the left leg; frequent headache and vomiting; loss of sphincter control; later complete paraplegia (22 years)	Complete paralysis of the lower extremities; weakness and atrophy of the muscles of the left arm; bilateral ankle and patellar clonus; Babinski sign on the right; sensory loss below D-1. Diagnosis: syringomyelia later spinal cord tumor	Laminectomy and resection of mass of veins at C-7 Death	Tortuous mass of varicose veins on dorsal and ventral surface of cord at C-7-D-3.
9 Benda	Age not given	Pains in both legs	Short period after onset, paralysis of the lower extremities set in (4 years)	Paraplegia. Diagnosis: multiple sclerosis	Death	Mass of varicose veins at L-1 and conus medullaris.
10 Loewenstein	Age and sex not given	Weakness of the lower extremities	Paralysis of both lower extremities	Paraplegia; sensory level at D-11. Diagnosis: "extramedullary lesion"	Laminectomy and partial removal of mass veins Death	Varicosities extended from D-4 to D-11. Corresponding cord degeneration.

TABLE I.—DILATATIONS OF SPINAL VEINS, WITH COMPRESSION OR OTHER INVOLVEMENT OF THE

TABLE I.—DILATATIONS OF SPINAL VEINS, WITH COMPRESSION OR OTHER INVOLVEMENT OF THE SPINAL CORD—Continued

Case No. Author	Age sex	Onset and early manifestations	Late manifestations and their duration	Neurologic findings and diagnosis	Course and termination	Anatomical findings
11 Alexander	40 M	Pain in the right ischial region	Pain increased in intensity (Few months)	Signs of compression of the right, first sacral root. Diagnosis: Compression of the root by tumor or meningitis	Laminectomy and removal of a dural varix Death (from thrombus)	Postmortem examination of the spinal cord showed no abnormality.
12 Alexander	37 M	Numbness of the right buttock, followed by weakness of the right leg and mild urinary incontinence	Slow progression with occasional remissions	Scar of old angioma of the neck (angioma had been removed in childhood). Angioma on the back; absent right ankle-jerk; analgesia in S-2-4 on right and S-4 and 5 on the left side. Diagnosis: angioma venosum racemosum of spinal cord	Laminectomy and mass of varicose veins found at D-10 Death (from paralytic ileus)	Varicose veins at D-10.
13 Spiller and Frazier	25 F	At 17 sudden attack of pain in the lower extremities; followed by numbness and weakness of the legs. Loss of sphincter control	Regained power in the legs a few months later, but 8 years later her right leg become suddenly weak and in 2 months lost power in her legs (8 years)	Spastic paralysis of the right leg; flaccid paralysis of the right foot; bilateral Babinski sign; exaggerated knee-jerks; impaired sphincteric control	Laminectomy and mass of varicose veins found at L-1 Improved	Tortuous varicose veins at L-1.
14 Blahd	14 M	Sudden but short attack of pain in the right groin; one month later another similar attack	Four months later, after another similar attack he became paralyzed in both legs and lost sphincteric control (6 months)	Flaccid paraplegia; absent abdominal reflexes; absent patellar and knee-jerks; loss of all forms of sensation below the crest of the ilium. Diagnosis: lesion at D-12	Laminectomy and mass of varicose veins found at level D-12 Unimproved	Convoluted varicose veins at D-12.
15 Muehsam	37 M	At age of 34 began to feel a sense of "heaviness" in the legs	Progressive weakness and numbness in both legs; urinary incontinence (2 years)	Spastic paraparesis; absent abdominal reflexes; bilateral ankle clonus and Babinski sign; sensory loss below the umbilicus. Diagnosis: syringomyelia later extramedullary tumor	Laminectomy and mass of varicose veins found at D-7 Death	Varices pia spinalis. Degenerative changes in the spinal cord.
16 Frazier and Russel	50 M	At age of 44 a feeling of "heaviness" and pain in the left leg set in. Pain spread to sacrum	Paræsthesias in the right leg; progressive paralysis of both legs; stiffness in the legs; later numbness in both legs (6 years)	Flaccid paraplegia; absent lower abdominal reflexes; hyperactive ankle and knee-jerks; bilateral Babinski and ankle clonus; hypalgesia below the umbilicus; loss of sphincteric control	Laminectomy and mass of varicose veins found at D-7-10 Improved	Convoluted varicose veins D-7 to D-10.
17 Sargent	31 M	At age of 29 sudden attack of sharp pain in the back on moving a ladder. His back became stiff	Pain and stiffness disappeared after 3 months, to reappear later and remain permanently; pain also radiated down the legs; stiffness in the left leg	No neurologic findings. Spinal fluid showed xanthochromia and high protein content. Diagnosis: spinal cord lesion	Laminectomy and mass of varicose veins found at L-1 Recovery	Tortuous varicose veins at L-1.
18 Sargent	52 M	At the age of 48 he developed pain, paræsthesias and weakness of the left leg. Numbness in perineal region. Anti-luetic therapy (4+ Wassermann)	Following temporary improvement, symptoms reappeared with paræsthesias first in the left leg and later in right. Numbness spread up to umbilicus (3 years)	Spastic paraparesis; absent lower abdominal reflexes; absent right knee and both ankle-jerks; bilateral Babinski sign; analgesis below the umbilicus	Laminectomy and a mass of varicose veins found at D-6. Not removed Improved (return of sphincter control).	
19 Sargent	38 M	Following a fall from a horse developed paræsthesia in the right leg. Pain disappeared after a few weeks	Pain in both legs; repeated attacks of weakness in the right leg; rectal and vesical disturbances. (7 years)	Paraparesis and atrophy of the legs; absent right knee and both ankle jerks; bilateral Babinski sign; sensory impairment in all sacral segments. Spinal fluid showed pleocytosis and increased globulin	Laminectomy and a diffuse "hæmangioma" found at D-10 to L-2 Unimproved.	
20 Sargent	53 M	Was struck on his back by a falling tree and lost power in his right leg. Regained power within 3 weeks	Intense pain in both legs which disappeared to reappear 3 months later. Weakness and wasting of the right leg; bowel and bladder disturbances; marked decline in condition (2 years)	Weakness of both legs; flabby atrophic muscles in both thighs; absent lower abdominal reflexes; absent ankle jerks; bilateral Babinski sign; sensory loss below D-10	Lamin ectomy and a mass of varicose veins found in lower dorsal segments Improved.	
21 Ritter	18 M	At the age of 16½ he developed weakness, cramp-like pains and paræsthesias in his legs	Urinary difficulty; progressive loss of power in both legs (1½ years)	Paraplegia; absent abdominal reflexes; absent ankle jerks; saddle-form anæsthesia. Diagnosis: tumor in conus medullaris or cauda equina	Laminectomy over sacral region—nothing found. Second laminectomy and mass of tortuous veins found at D-10 Improved markedly.	

TABLE I.—DILATATIONS OF SPINAL VEINS, WITH COMPRESSION OR OTHER INVOLVEMENT OF THE SPINAL CORD—Continued

Case No. Author	Age sex	Onset and early manifestations	Late manifestations and their duration	Neurologic findings and diagnosis	Course and termination	Anatomical findings
22 Ritter	47 M	A feeling of "tiredness" set in his legs. Collapsed while at work 7 months later	Recovered after 2 months but weakness in his legs continued to increase; cramp-like pains in legs and sacrum; urinary retention (1½ years)	Paraplegia; atrophy of the leg muscles; absent abdominal reflexes; absent knee and ankle jerks; all forms of sensation lost below L-1; lipiodol test was negative. Diagnosis: tumor of the spinal cord at D-8-12	Laminectomy and a large varicose vein thrown into a mass of coils at D-7. Removal of vein and electrocoagulation Death	Postmortem examination showed epidural inflammatory changes fresh degeneration of cord.
23 Rand	28 F	At the age of 27 she had 2 falls and shortly after developed pain in the right thigh. Later spastic contractures also	Following a remission of 4 months the pain returned with greater severity and after a sneezing paroxysm she developed paralysis and numbness in both legs (1 year)	Paraparesis; atrophy of the right thigh with right foot drop; saddle form hypæsthesia; parasthesia in the right leg; absent right ankle jerk; urinary retention and rectal incontinence. Diagnosis: tumor at S-3-5.	Laminectomy and a varix of veins found at L-2 to D-12 Improved markedly (power returned).	
24 Rand	30 M	At age of 27 he began to have pain in the left foot and shortly after weakness of the left foot	Within a year developed left foot drop and atrophy of muscles of left leg; after a stationary period of a year he suddenly became paralyzed in both legs and lost control of bowel and bladder (3 years)	Flaccid paraplegia; absent knee and ankle jerks; incontinence of bowel and bladder anesthesia in all forms below L-2; dark pigmented patch under the right scapula; spinal fluid xanthochromic and positive Frein test. Diagnosis: tumor or cyst of cord—skin nævous suggested an hæmangioma	Laminectomy and a mass of varicose veins found at L-2 to D-11 Unimproved.	

much dilated vessels with poorly defined vessel wall. The cord was disorganized. Below the aneurism, in the lumbar cord, was a descending degeneration of the left lateral pyramidal tract and to a smaller degree the same tract on the right side. In the dorsal segments of the cord was added also degeneration of the posterior columns and a mild degree of degeneration in the direct cerebellar tract.

Though lues was excluded as an etiological factor, evidence of a subacute inflammatory process was present in the periphery of the aneurism. The cause of this inflammation is not known. The possibility of its being an end-result of a subsiding infection is suggested. Thus periarteritis is believed to be a factor in the production of the aneurism. Of additional interest in this case is the absence of the lateral spinal arteries.

Elsberg's case (18) a boy aged 13 years, was well up to the age of 11, when an accidental fall resulted in a condition which was considered as "concussion" of the spine and which confined the patient to bed for 2 days. A year later he began to complain of pain in the left thigh and soon after of pain in the right leg. As the pain abated, progressive weakness of the lower extremities set in and was followed somewhat later by bowel and bladder incontinence.

Examination revealed spasticity in both lower extremities, exaggerated knee jerks, bilateral ankle clonus, bilateral Babinski sign, and absent abdominal and cremasteric reflexes. There was hypæsthesia from the second lumbar to the first sacral. A large flat lipoma was also noted over the sacral region.

A laminectomy was performed. On reflecting the dura, in the region of eighth to eleventh dorsal vertebræ, a mass of tortuous blood vessels came into view (Fig. 12.) This mass measured 4 centimeters in length. A vein and artery were noted entering this mass from above, and two large vessels could be traced from the lower end to the cauda equina. A few branches entered the mass from the anterior part of the cord. A part of the mass was removed without change in the neurologic condition.

This is the only case of its kind and is regarded by the author as a true arteriovenous aneurism of the dorsal spinal vessels, which he believes was the result of the trauma which the patient received 2 years previously. Of interest here is the observation by Kadyi who found in the white matter of a spinal cord similar intercommunication between artery and vein.

Balo's case a male, aged 63 years, suddenly developed pains in the perineal region, attacks of

severe pains in the abdomen, difficulty in urination and defæcation, and diminution of power in his legs. Examination revealed inequality and fixation of pupils to light; absent right knee jerks; exaggerated left knee jerks. Diagnosis of tabes dorsalis was made. He died shortly after admission, of pneumonia.

Autopsy findings. In the region of the third to fourth lumbar vertebræ, there was a small growth covered with blood and compressing the spinal cord. The veins in that region of the growth were found to be enlarged. The growth was found to be microscopically an arterial aneurism.

The aneurism between the pia and arachnoid as pointed out by the author apparently sprang from the branch of the posterior spinal artery which comes off from the dorsal trunk of the intercostal artery. In considering the possible etiology of the aneurism, lues is given as the most likely cause. Although the patient denied syphilis, the luetic aortitis found at autopsy is strong evidence of the presence of the disease. The subarachnoid hæmorrhage may be taken into consideration as the cause of death. It also throws some light on the etiology of subarachnoid hæmorrhage being due to aneurisms.

Sargent's case (18), a male, 44 years of age, was well up to the age of 42, when he began to develop increasing weakness in the right arm and wasting of the muscles of the right hand. He also had pain in the region of the right shoulder. Later he began to lose power in his lower extremities and developed urinary incontinence.

Examination showed loss of power in his legs, profound sensory loss up to level of first dorsal, urinary retention, absent knee jerks, active ankle jerks, bilateral Babinski. Spinal fluid showed an increased protein content and negative Wassermann.

Laminectomy was attempted but was abandoned because of excessive bleeding. The muscles and bones were permeated with numerous dilated, tortuous, thin-walled arteries. The condition was that of diffuse aneurismal varix. Patient died shortly after operation.

Autopsy disclosed the presence of an aneurism similar to that seen in the muscles, at the level of seventh cervical, containing a recent clot. The cord was markedly compressed. Section of the cord showed the intramedullary vessels of normal size and structure.

Elsberg's case (22) male, aged 54, began to have pain in the back of his neck 1½ years before operation which was followed shortly after by paræsthesias and numbness in his fingers. He also rapidly lost power in his upper extremities. A year later he began to lose power and suffer pain in his legs. He was bedridden for 3 months and lost control of his sphincters, for about the same length of time.

Examination showed spastic paralysis of the upper extremities, exaggerated deep reflexes in the upper and lower extremities, paresis of the lower extremities, absent abdominals, bilateral Babinski and clonus: sensation lost in all forms below the second cervical. X-ray examination was negative.

The diagnosis was neoplasm within the foramen magnum, extending into the cord. Laminectomy including the first to fourth cervical vertebræ was done, no tumor was found but when the probe was passed upward an obstruction was encountered above. Patient died 2 days later from respiratory paralysis. Postmortem examination showed a large aneurism of the right vertebral artery, measuring 3 by 4 centimeters; the medulla and cord from the first to the third cervical vertebræ were markedly compressed.

Aneurisms of vertebral arteries are not very uncommon and belong rather to the intracranial type of lesion, but in the presence of clinical features pointing to a high cervical lesion this case may be included in this group.

Heboldt's patient, a girl 15 years of age, was well until 9 months before death. Then in the course of a septic infection (erysipelas of the face) she developed a wide-spread disease of the brain with such manifestations, as impaired hearing, fixation of pupils, ptosis of eyelids, athasia, dementia, disturbances in reflexes, indicating a progressive encephalitic process.

The autopsy disclosed diffuse meningitis, thrombosis of the left sinus transversus, multiple abscesses of the brain, myelitis and an aneurismal formation of vessels, alongside some capillaries showing thrombotic changes and an occasional small capillary hæmorrhage.

Heboldt raised the question *whether these vessels changes in the cord were congenital* or *acquired*. The presence of thrombosed spinal cord veins, however, suggested to him the possibility that they may have caused the dilatation of the venous capillaries and the aneurismal sac formations with subsequent rupture and hæmorrhage. But the structure of the aneurismal formations themselves appeared to be more in the nature of a developmental congenital condition. It is quite possible that this vessel anomaly is developmental in origin, without any bearing on the clinical manifestation in this case and is but an incidental finding.

GROUP III. HÆMANGIOMA

The hæmangioma is commonly defined as a tumor composed of newly-formed vessels

TABLE II.—INTRAMEDULLARY HÆMANGIOMATA

Case No. Author	Age Sex	Early Symptoms	Late Symptoms Duration	Neurologic findings Diagnosis	Course Termination	Nature of tumor
1 Glazer	42 F	Sudden onset of lancinating pain in the legs during late months of pregnancy	Paraplegia at the end of pregnancy; after short period of improvement, she developed stiffness in the legs, ataxia, sensory loss below umbilicus (2 years)	Paraparesis; paræsthesia and incoordination in the legs; sensory impairment below the umbilicus; atrophy of the leg muscles and urinary disturbances	Gradual decline decubitus ulcers pyelitis sepsis Death	Central angiosarcoma of the entire spinal cord.
2 Gaupp	?	?	?	?	Death	Large cavernous angioma below conus medullaris.
3 Berenbuch	15 M	Sudden onset of weakness in the legs, following violent exercise	Lancinating pain in the legs; complete paralysis of the right and partial paralysis of the left leg (2 months)	Paralysis of the right and weakness of the left leg; exaggerated deep reflexes; hypalgesia below the umbilicus; multiple subcutaneous lipomata present since birth. Diagnosis: intraspinal lipoma	Laminectomy Death	Intramedullary hæmangioma (also intraspinal angioma of the dura and large cavernous plexus along the spinal column).
4 Lorenz	27 F	Injury to the back of the head; headaches for several days. Recovery	Eighteen months later, sudden onset of weakness in the left hand followed by collapse (2 days)	Paralysis of both legs	Death occurred one day after the collapse	Cavernous angioma left lateral column (hæmorrhage in cervical and dorsal segments).
5 Schultze	29 M	Gradual onset of dizziness and pain in the left arm	Girdle phenomena; pain in the spine; weakness in the left leg (15 months)	Paresis and atrophy of the left arm; paresis of the left leg; exaggerated knee and ankle jerks; Babinski sign on the left; sensory impairment on the right to the upper dorsal level. Diagnosis: intramedullary or extramedullary tumor	Laminectomy and enucleation of tumor, at D. Recovery	Intramedullary angioma.
6 Roman	32 M	Girdle phenomena and constipation following an attack of influenza	Weakness in the lower extremities; incontinence of stool; bedridden with complete paralysis (2 years)	Paraplegia; exaggerated knee and ankle jerks; sensory loss below the umbilicus; total incontinence and decubitus ulcers. Diagnosis: tumor of the spinal cord	Sepsis Death	Intramedullary angioma at D-8 to D-11.
7 Pinner	Age not given M	Gradual onset of numbness in the hands, which spread to the arms	Weakness in the arms and legs (1 year)	Paresis of all four extremities; atrophy of the arm muscles; exaggerated deep reflexes; Babinski sign on the left; diminished sensation in all four extremities	Gradual decline during the next year developed complete paralysis of all extremities Death	Intramedullary angioma (filling the interior of the cord and containing a syringomyelic cavity in the center).
8 Henneburg	58 M	Pain in the left knee and shoulder	Weakness in the left arm and leg; numbness and ascending pain in the left leg	Atrophy of the left hand and the right arm; paræsthesias in both legs. Absent knee and ankle jerks. Cerebrospinal fluid negative	Paraplegia; weakness of arms; reappearance of reflexes. Decubitus ulcers. Bronchopneumonia Death	Intramedullary angioma at C-4 to C-8.

TABLE III.—EXTRAMEDULLARY PIAL HÆMANGIOMA

Case No. Author	Age Sex	Early Symptoms	Late Symptoms Duration	Neurologic findings Diagnosis	Course Termination	Nature of tumor
1 Harman and Balck	Age not given M	Pain in both legs; 4 months later sudden increase in intensity of pain with loss of power in legs. Opisthotonus	Gradual decrease in signs with complete disappearance in 6 months; second similar attack 2 years later of short duration and complete recovery; following remission of 2½ years, recurrence of weakness and severe pain in the legs (Total duration 5½ years)	Retraction of the neck; paraparesis; dysphagia; dysarthria; bowel and bladder incontinence. Diagnosis not stated	Rapid decline. Sepsis, coma Death	Large blood clot compressing lumbar segments of cord; blood arising from angiomatous mass on dorsal surface of cord. Hæmorrhage confined to subarachnoid space and extended upward to cervical enlargement.
2 Hadlich	35 F	None referable to intraspinal lesion	None referable to intraspinal lesion	None recorded	Dwarfed patient underwent a cesarean section because of small pelvis. Operation refused in Death	Angioma of pia in lumbar segments.
3 Cobb	8 M	While playing suddenly seized with severe pain in lower part of back; felt weak and was nauseated	Pain subsided but returned in a week; in addition there was numbness in the left foot which spread up to involve the entire leg; within 24 hours he was completely paralyzed in both legs and had vesical and rectal incontinence (8 days)	Complete flaccid paraplegia; absent knee jerks and abdominal reflexes; analgesia below umbilicus; cerebrospinal fluid contained 20 cells per c.mm. Colored birth mark was noted on back, but no significance was ascribed to it at first. Diagnosis: poliomyelitis	Condition and diagnosis remained unchanged for 6 weeks, when paralysis became spastic and reflexes began to return; bilateral Babinski sign appeared. Dr. Cushing finding nævus in same metamere as level of supposed cord lesion suggested the diagnosis of dermoid or hæmangioma. Laminectomy performed; tumor not removed. Patient not improved	A tangle of pulsating vessels, underneath the dura, filled the spinal canal. The cord was flattened.

(Ewing). This term, however, is somewhat more restricted by Borst, who would under this name consider only such tumors, which bear the characteristics of a new-growth and in which an angioblastic process is in progress. He would exclude from this group such vascular tumor-formations as the telengiectases and the cavernomata, which he regards as congenital malformations rather than true new-growths. Nevertheless, it is still good practice to include these forms of vessel anomalies among the hæmangiomata. They are usually classified as simple and cavernous.

The simple hæmangiomata are of no interest to us here, for they occur most commonly in the skin, as nævi or telangiectasis, or in muscles and seldom if ever in the central nervous system. They are composed of many capillary vessels and are more in the nature of a malformation than a new-growth.

The cavernous angioma or cavernoma has the liver and spleen as the location of predilection, but is not infrequently found in various parts of the central nervous system and their enveloping structures. Because of their effect on the spinal cord, their occurrence in the vertebral column and the epidural space is of particular significance here.

Histologically, such a tumor consists of widely-dilated vascular channels, which are separated by a variable-in-amount connective tissue. The tumor is usually encapsulated and is benign in character, eroding adjacent tissues but not invading them. It is very often multiple, occurring in several systems, or several divisions of a given system. The frequent occurrence of primary multiple angiomata, with multiple foci in several parts of the same system, as well as the not uncommon finding of active blood formation in some of the angiomata of the liver, speak in favor of their congenital and embryonal "rest" character. The tumor may occasionally assume a malignant character, but then it falls more properly into the group of the so-called hæmangio-endothelioma.

These intraspinal hæmangiomata are assembled in the third group of our material. They show certain variations in their relationship to the walls and contents of the spinal canal so that it is found convenient to classify them

TABLE IV.—EPIDURAL HÆMANGIOMATA

Case No. Author	Age Sex	Early Symptoms	Late Symptoms Duration	Neurologic findings Diagnosis	Course Termination	Nature of tumor
1 Gerhardt	23 F	Weakness right leg and later weakness of the left; cramp-like pains in legs	Paraplegia; anæsthesia to knees (2 years)	Paraplegia; anæsthesia to umbilicus; exaggerated deep reflexes; loss of sphincter control. Diagnosis not given	Stationary for 3 years, then sepsis set in, followed by Death	Angioma between dura and bone at D-6.
2 Devic and Tolot	37 F	Pain in right arm, hypertrophy of the arm simulating elephantiasis. Amputation of the arm	A tumor appeared in the left breast; pains in the legs and paraplegia spastica (4 years)	Syringomyelic sensory changes; paraplegia changed from spastic to flaccid type. Diagnosis not given	Rapid decline and Death	Multiple epidural angiomata at several levels.
3 Saltykow	28 F	Sudden onset of paralysis of the lower extremities	Complete paraplegia (3 weeks)	Paraplegia. Diagnosis: Pott's disease	Death	Multiple epidural angiomata.
4 Beriel	41 F	Progressive weakness in the legs	Severe cramp pains in legs; difficulty in walking (2½ years)	Spastic contractures in the legs; spasm of the abdominal and lumbar muscles; bilateral Babinski sign, sensory loss below D-6. Diagnosis: intraspinal tumor	Laminectomy and removal of extradural hæmangioma. Improved	Extradural vascular collections at D-4 and D-5.
5 Hille	57 M	Paræsthesias in his feet	Muscular twitchings and later paraplegia; bowel and bladder disturbances (7 years)	Paraplegia; bilateral Babinski sign; absent abdominal reflexes; exaggerated deep reflexes; sensory loss below D-4. Diagnosis: extramedullary tumor	Laminectomy and removal of tumor. Recovery	Extradural angioma D-7 to C-7.
6 Elsberg (23)	64 M	Pain in left lower chest	Pain and weakness in both legs; abdominal girdle sensations; urinary difficulty (1 year)	Spastic paraparesis; bilateral Babinski sign; absent abdominals; bilateral ankle clonus; hypæsthesia below D-6; anæsthesia below L-3. Diagnosis: extramedullary tumor	Laminectomy and removal of tumor Recovery	Extradural angioma D-2 to D-5.
7 Gold	23 M	Weakness of both legs	Paraplegia; occasional paræsthesias in the legs (5 years)	Paraplegia; bilateral Babinski sign; deep reflexes exaggerated; tenderness over D-6. X-ray showed decalcification of D-6 and 7. Diagnosis: intraspinal tumor	Laminectomy and removal of tumor. Operation terminated in Death	Extradural cavernous angioma (process also in D-6 vertebra).
8 Perman	24 F	Pain in legs; weakness in knees	Weakness in right leg; temporary improvement; weakness also numbness in right leg returned (2 years)	Spastic ataxic gait; later paraplegia; bilateral ankle clonus and Babinski sign; loss of sensation below D-10. Diagnosis: tumor of the cord	Laminectomy and removal of tumor. Recovery	Extradural angioma growing from D-8 vertebra.
9 Elsberg (23)	23 M	Gradual onset of numbness and a feeling of "cold" in both feet. Weakness in the lower extremities	Progressive weakness in both legs, with remissions; difficulty in walking; tremors in legs (1½ years)	Spastic paraparesis; bilateral patellar clonus; right ankle clonus; bilateral Babinski sign; hypalgesia over inner lower part of the right leg; thermanæsthesia below D-3. Diagnosis: neoplasm at D-3	Manometric readings of cerebrospinal fluid showed a complete block. Laminectomy and removal of an extradural tumor	Extradural capillary hæmangioma.
10 Belado and Morea	36 F	Rigidity and pain in lower extremities setting in during pregnancy. Weakness in the legs set in shortly after	She recovered fully following delivery; she had recurrences of these attacks with recovery during the next 3 pregnancies; the 5th pregnancy showed paralysis of the right leg only; in her 7th and 8th pregnancies she had incontinence with paraplegia (10 years)	Spastic paraplegia; absent abdominals; bilateral Babinski sign; right ankle clonus; pain and temperature sense lost below D-12; diminished below D-8; proprioceptive sense lost in the legs; incontinence of bowel and bladder; decubitus ulcers; gravid uterus. Diagnosis: intraspinal tumor with recurrent bleeding	Queckenstedt test showed complete block. Died during labor from cardiac failure	Extradural hæmangioma at D-3 to D-7.

TABLE V.—VERTEBRAL HÆMANGIOMATA

Case No. Author	Age Sex	Clinical manifestations	Duration of symptoms	Neurologic findings Diagnosis	Autopsy findings
1 Virchow	Very old F				Two hazelnut-sized angiomata in two separate vertebræ.
2 Deetz	42 F			Signs of compression myelitis. Diagnosis: sarcoma of vertebræ	Angioma of spinal vertebræ.
3 Muthmann	61 F	At first left-sided sciatica, which later affected the other side. As pain lessened weakness set in both of the legs	16 years	Paraplegia. Diagnosis: spondylitis	Cavernous angioma compressing the spinal cord at D-6.
4 Ribbert	Very young F			Signs of compression myelitis	Cavernous angioma had destroyed the vertebra and was compressing the cord.
5 Pentman	56 F	Abdominal pains	8 years		Multiple angiomata (one in L-1 vertebra).
6 Trommer	17 F	Sudden onset of weakness in the legs, soon followed by complete paralysis of the legs; numbness below the umbilicus; pain in back and ribs	3 weeks	Paraplegia; contractures in legs; tenderness over D-6 vertebra; decubitus ulcers. Diagnosis: tumor of the spinal cord	Angioma in thoracic vertebra, compressing the cord and extending into the epidural space.
7 A. G., Mt. Sinai, No. 133434 10-12-1912	13 F	One week following a fall developed weakness and stiffness in legs; bladder incontinence and loss of sphincteric control of rectum	1 year	Spastic paraplegia; absent abdominal reflexes; bilateral ankle clonus; bilateral Babinski; sensory loss below D-10. Bladder and rectal control impaired. Laminectomy and severe hæmorrhage from an aneurismal vessel. Death	Angioma of eighth dorsal vertebra, small epidural hæmangioma extending into spinal canal, and compressive myelitis (Fig. 15).

as follows: Group III a, intramedullary; Group III b, extramedullary (pial); Group III c, dural; Group III d, vertebral.

Group III a—Intramedullary hæmangioma (*Table II.*) This sub-group consists of but few cases, as hæmangiomata in this location are apparently rare. They are also marked by a variability in the character of the initial signs and symptoms and lack of uniformity in the unfolding of the clinical picture. In only one case was surgery of a distinct service.

Group III b—Extramedullary (pial) hæmangioma. (*Table III.*) This group consists of only 3 cases. Of interest here is the case of Cobb, in which the pre-operative diagnosis was aided by the finding of a nævus in a dermatoma corresponding to the sensory disturbances. The case of Harman and Balck also offers interesting clinical features. The repeated attacks, the remissions, the appearance of signs of meningeal irritation in the final episode are highly suggestive of the character of the lesion.

Group III c—Epidural hæmangioma. (*Table IV.*) This, the largest group of hæmangiomata, is of particular interest, for it is in this location that this type of intraspinal vascular tumor promises most in surgical intervention. In 5 of a total of 10 cases, laminectomy and removal of tumor resulted in partial or complete recovery. In one case laminectomy was followed by death. In the other 4 cases laminectomy was not at all attempted.

In the majority of instances, the clinical features are such as to indicate an expanding intraspinal lesion and to point most commonly to an extramedullary location. There are, however, no signs for a pre-operative identification of the character of the lesion. Still the indications for operative interference are quite clear particularly in view of the fair outlook for the removal of the tumor with either total or partial recovery.

Group III d—Vertebral hæmangiomata. (*Table V.*) It is commonly said that hæmangioma in the vertebral column is a rare occurrence (Kaufman, Aschoff, and others) but, in a recent contribution, Makrycostas described 12 cases of vertebral hæmangioma, which he was able to collect in the routine postmortem work in Erdheim's laboratory.

He is of the opinion that such localization of hæmangiomata is not among the rarest. He ascribes the scarcity of such material in the literature to the infrequency with which signs and symptoms of spinal cord compression accompany vertebral hæmangioma. This is true of his 12 cases in which no neurological manifestations were recorded and hence their clinical histories are not included in Table V.

Makrycostas made the following important observations: (1) vertebral hæmangiomata are most commonly found in late adult life; (2) hæmangiomata are seldom unilocular, they are most frequently multiple; (3) hæmangiomata are very irregular in their distribution but are most frequently found in the lower dorsal and lumbar vertebræ; (4) vertebral hæmangiomata are not infrequently associated with co-existing epidural vascular tumors, the latter being most often responsible for the manifestations of cord compression. The last statement finds support in Elsberg's case (Table V, case 7) in which an epidural hæmangioma was found alongside a vertebral vascular tumor (Fig. 15).

The available clinical histories of the individual cases in this group, with the exception of Trommer's case (Table V), are too incomplete to be of service. Because of the nature of the lesion and its tendency to multiplicity, little, of course, may be expected from operative interference. From the material so far recorded, no conclusions may be drawn as to whether X-ray examination of spinal column may be of diagnostic aid. There are on record only two cases in which X-ray examination of the spine was done. In Gold's case the X-ray film revealed decalcification of the involved vertebra. In the case of Permon, however, antimortem X-ray examination failed to disclose any destructive process in the spinal column, while a postmortem preparation revealed marked thinning of the bony tissue in the involved vertebra.

GENERAL SUMMARY AND CONCLUSIONS

In spite of the oft repeated statement, that aneurismal dilatations, or so-called venous angiomata of intraspinal vessels, are exceedingly rare, and contrary to the belief expressed by Bruns that when they do occur they are not of such a size as to produce clinical signs, we have presented here 28 verified cases illustrating this variety of intraspinal vessel alteration with signs and symptoms of cord compression. Moreover, the above number does not exhaust all of the reported cases, as other similar instances are described by Kadyi, Lennep, Elsberg (21), Sick, Adson, and Dandy, but are not included in this survey, since they were merely mentioned in statistical studies and were not accompanied by detailed clinical descriptions. The total number of recorded instances is certainly sufficiently impressive to discount the old belief that such pathological alterations of intraspinal vessels are unusually rare.

We have already said elsewhere that the cases in Group I, reveal no definite signs or symptoms, which could aid in the clinical identification of the true character of the lesion. In the large majority of instances they simulate clinically very closely extramedullary tumors including even the irritative root phenomena, but differ from them in that they more frequently show atypical manifestations because of the dissemination of the lesion or its invasion of the cord substance and in that no subarachnoid block may be disclosed by the manometric or lipiodol tests. Thus, given a case with signs and symptoms of cord compression with or without atypical features of direct cord involvement, with no demonstrable subarachnoid block occurring, in an individual in his late adult life, a lesion in the nature of venous dilatation of intraspinal vessel may be considered among and alongside of other diagnostic possibilities. In any event, an exploratory laminectomy is indicated. It is, however, necessary to bear in mind, that instances are not infrequent in which extensive areas of the white and gray matter of the spinal cord are invaded by such dilated vessels and have undergone degenerative changes. Hence, if an exploratory laminectomy discloses dilated veins on the surface of the cord, thorough investigation of the spinal canal is required to ascertain that there is no extension of the lesion into the substance of the cord and to exclude the possible existence of a spinal cord neoplasm at a somewhat higher

level. In the event that extension of venous alteration into the substance of the cord is found, decompression is all that should be undertaken, for more radical steps, such as removal or partial resection of the vessels may lead to fatal termination, while the decompression will likely give quite satisfactory results.

We can add little to what we have already said in the introductory remarks as to the anatomical features of the lesion. It was suggested that two factors: a degenerative lesion in the vessels (phlebosclerosis) of more or less generalized character and a precipitating cause, such as unusual posture, inflammatory process (meningitis) excessive muscular activity, trauma or any other factor which will impede the return flow of blood, must combine in producing such anatomical alterations. The latter is not to be confused with true angioblastic lesions.

Group II, which consists of the arterial or arteriovenous aneurism of intraspinal vessels, is from the clinical point of view a less satisfactory category. All that can be said is that clinically the individual members fall best into Group I, while on purely morphological grounds they must be separated.

In Group III with all its subdivisions we have assembled only those cases, in which the anatomical features justify the use of the term "hæmangioma." It is obvious then that the term "angioma or hæmangioma" as in this contribution has been restricted to true vascular tumors, though it is a rather common practice to include incorrectly under the term "angioma" conditions which are best described as "venous dilatations." The existing confusion in the terminology is in part responsible for this survey and has prompted us to review all available instances of true hæmangiomata and incorporate them in this article. It was thought to be highly desirable and timely to differentiate these two conditions and put an end to the chaotic classification of such material. Clinically the hæmangiomata, unlike the venous dilatations, are more apt to give rise to very discrete signs of cord compression. A clinical differentiation between hæmangiomata of various localizations is difficult if not impossible, with our present state of knowledge, though the opportunity offered by X-ray examination in instances in which bone rarefication may be apparent in vertebral hæmangioma should be utilized. With negative X-ray findings, laminectomy is indicated for if an angioma of the extramedullary or extradural type is found the promise for removal and permanent cure is very good. One must bear in mind, however, that the vertebral hæmangiomata are likely to give trouble by bleeding.

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CONTRIBUTING CAUSES OF GENITO-URINARY ANOMALIES[1]

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ANOMALIES occur not only in animal life but in all kinds of cell life. The anomalies of animals usually lead to death, since no one has the desire to interfere with such natural laws, except in case of domesticated animals for show purposes. Man is subject not only to the anomalous conditions which are found in the lower animals but to those connected with the higher nervous system with which he is endowed. The weight of the nervous system is half that of the embryo at three months.

One of the most serious of developmental defects is that of exstrophy of the bladder. This is sometimes associated with other defects, some of which may greatly complicate the condition.

The anomalies occurring at the caudal end of the body are of clinical interest. In early fetal life the developing bladder and rectum are one. The anterior portion of the cloacal cavity consists of the allantois and wolffian ducts, from which are developed the sex organs and the urinary collecting system.

Mammalian embryos may be divided into two groups: those which retain functional wolffian bodies until the kidneys are sufficiently developed to excrete urine, as in birds and reptiles, and those in which the wolffian bodies degenerate before the kidneys reach functional ability. The first group includes the pig, the sheep, and the cat; the second, the rabbit, the guinea pig, man, and the rat.

The allantois is the receptacle of the urine formed within the body of the embryo; it is present as a reservoir only in animals with embryonic excretion, and its size varies with the size of the wolffian bodies and with their stage of development.

The embryonic and fetal urinary excretion takes place wholly through the placenta in the rat; in the rabbit, guinea pig, and man it is first through the wolffian body and later through the placenta, but never through the placenta in the pig, sheep, and cat.

The kidney-secreting tissue extends as mesothelial bodies or nephrogenic tissue from the lower dorsal vertebra down to the second sacral. They lie close together with the aorta between. This substance is supplied by many blood vessels derived from a delicate plexus surrounding and connected with the aorta. The ureter and pelvis of the kidney develop from a pouch which early appears from the lower portion of the wolffian duct. This collecting portion becomes attached to the secreting portion by climbing up the ladder of the blood supply, so to speak, of the nephrogenic substance. The numerous blood vessels atrophy as the pelvis of the kidney ascends to its higher position, and the secreting substance arranges itself over it and forms a capsule. The two mesothelial bodies may touch each other and become fused, developing the horse-shoe kidney or various attachments to each other; 90 per cent of the horse-shoe kidneys are fused at the lower pole. Some of the mesothelial or secreting portion of the kidney may not become connected with the collecting portion and may then retain its embryonic type, forming a mesothelial rest from which may develop so-called hypernephroma or, more correctly, mesothelioma of the kidney. In other cases failure of connection between the secreting portion with the collecting cavity and continuance of secretion without elimination, form congenital cystic kidneys, usually double, with one large cyst or multiple cysts in each.

Wherever the kidney stops in the process of union of collecting and secreting portions, its renal artery develops from the major artery supplying it at the time. This is the lower of the upper group of five arteries or the upper of the middle group of five arteries. As growth continues, the delicate vascular plexus outside the aorta disappears and the renal artery comes directly from the aorta, but owing to change in position with development it may come from a lower position on the aorta, from the sacral artery, or from the

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common iliac. Malposition of the kidney is not so serious if function is not disturbed, but it may lead to injury. Excessive mobility is not a disease unless renal function is interfered with or the kidney in its movements disturbs some other organ; thus a movable right kidney may disturb a diseased appendix, the appendix, however, being the primary offender. Mobility may interfere with delivery of urine by kinking the ureter over a band of connective tissue or an anomalous artery which is occasionally present and connects the lower pole of the kidney with the aorta; this is one of the original mesothelial vessels which failed to disappear and which, if occurring in the upper pole, would not cause harm. One kidney may be missing from failure of development of the mesothelium (the secreting structure) or its failure to connect with the collecting portion. Three or four kidneys may be present with an equal number of complete or partial ureters. A splitting of the collecting portion at the wolffian duct causes double ureters and fused or separated double kidneys on one or both sides. The division of the pelvis into several tubes connecting with one or two ureters is normal in the otter and beaver.

In rare cases the proctodeum surrounded by two muscle sphincters which should connect the skin with the rectum does not form, and in such cases it is not uncommon for the rectum to remain connected with the outlet of the bladder in the membranous portion of the urethra in the male or vagina in the female, a remnant of the cloaca.

The anal muscle may be weak normally or it may be weak because poorly innervated with rectal prolapse which sometimes occurs; it is most important to know this before any effort is made to transplant the ureters as the rectum must have good control at the outlet or the patient's condition will be worse than before.

Exstrophy of the bladder, then, should occur later than the separation of this cloaca in the rectum and bladder. In most cases of exstrophy, the remnant of the umbilicus scar shows at the upper margin of the exposed mucous membrane of the bladder. In a small number of cases the umbilicus is normal with good skin between it and the wall of the bladder. It is significant that the last muscle to develop is that covering the urinary bladder; in some cases there may be spots of deficiency—openings in the muscle, exposing the mucous membrane. Through these openings a full bladder pushes out the mucous membrane and small hernias develop which become small or large diverticula of the bladder, but not having any muscle they are never able to empty themselves into the bladder and are always kept full, increasing in size by tension of the bladder. They occasionally become infected and stones may develop within them. These openings vary in size; in some cases when the bladder is open they hardly show, in others they are large enough to admit a thumb. When the bladder is empty these diverticula remain as full as intra-abdominal pressure permits.

In exstrophy of the bladder, the pubic bones show as lateral short stumps and are not connected at the pubic arch; the urethra is split wide open above (epispadias); the bladder is also split from top to bottom and spreads out on the surface of the abdomen. It appears as if the bladder, before it was ready to retain urine, had been compelled to receive it and thus it bulged up between the developing pubic bones and prevented their union. Finally the whole sac splits from top to bottom, but for a considerable period it had been dilated from holding the urine. Other similar defects occur, such as spina bifida, caused by increased cerebrospinal fluid bulging the sac in the weaker places with less support and preventing the union of the spinal lamina which should cover the cord. Rarely the central groove fails to close as a tube and a condition called rachischisis results, which causes early death; the spinal tube being spread open appears as a mucous membrane. In rare cases the bladder is formed and completely covered with skin and the pubic arch has united, but the prostatic portion, the membranous portion, and the spongy portion of the urethra are completely split as in epispadias, and the skin on the upper side passes into the mucous membrane of the bladder; muscular tissue does not develop in the lower portion. Although the

surface of the bladder is not exposed to constant irritation by the absorbent cloths which are usually worn the condition should be regarded as true exstrophy, and the patient made at least dry and comfortable by transplantation of the ureters into the large bowel. That carcinoma sometimes develops from chronic irritation is well known; the wearing of absorbent cloths on the exposed bladder has caused cancer in six cases of complete exstrophy observed in the clinic. The ages of the patients varied from 22 to 46 years.

The experience of transplanting the ureters for exstrophy of the bladder has led to similar procedures in cancer of the base of the bladder, which is attendant with such constant suffering. In such cases we have transplanted the ureters and removed the bladder, to the great relief of the sufferers.

Until recently the public did not know that something could be done for such defects, but now patients of all ages are coming for consultation in the hope that something can be done. The chronic rubbing of the exposed surfaces results in fibrosis around the ureteral outlets, and older patients as a rule have greatly dilated ureters and hydronephrosis or pyonephrosis. In some cases a diagnosis is made by roentgen-ray examination after the ureters have been injected with something opaque which readily shows their size and the condition of the pelvis of the kidney. Some degree of inguinal hernia is common in exstrophy; probably in the male this is due to failure of attachment of the gubernaculum which should fix the testis and, as the body elongates and grows away from it, hold the testis in place for development in the scrotum. If the testes are undescended they become sterile and cystic. Sometimes the uterus is partly divided at the fundus or it may be completely divided, emptying into one cervix, or the uterus may be double with two cervices and vaginas. I have seen two cases of the latter condition.

Since the cloacal state is the natural one in fowls, it early came to mind, in the treatment of these cases, to divert the urinary flow, if possible, into the rectum.

The loss of full nerve control in this period of development might occur as a result of spina bifida occulta which is not infrequently associated with the anomaly of exstrophy. Secondary trouble from the traction on nerves might develop since the opening in the bone, usually at the upper sacral segment or lower lumbar segment, may so fix the cord structure by the attachment of the dura that the rapid overgrowth of the spinal column, previously the same length as the spinal cord, injures the pelvic nerve by tension. Some cases of enuresis are due to spina bifida occulta in this lower region reducing the strength of the normal automatic control.

In past ages, through evolution, many types of life came on earth that were found wanting in various particulars and disappeared, or some made further changes and in a different manner continued to exist; for example, the dinosaurs, with small heads and brains, long necks, and big bodies, cold-blooded egg-laying animals, disappeared. Nature and evolution dislike waste, and whenever possible they use tissues for other purposes when the type of structure and the form of life change.

Spina bifida is a possible complication in exstrophy of the bladder. It is of interest to note that where spinal fluid appears near the skin the hair grows. In the adult, then, a patch of hair in an area on the median line of the back probably means that it covers posterior spina bifida occulta, although there is no bulging in the area. It does not occur in the anterior type.

The change of the invertebrate to the vertebrate was a drastic one, as the cephalic stomach with its straight gut was behind the nervous system in the invertebrate and became changed to a position in front of the nervous system in the vertebrate.

In the human embryo, between the seventeenth and the twenty-second days, there are 3 days in which the large central tube of the spinal column, which at this time is larger than the large bowel, is connected with the large bowel at a small opening called the neuro-enteric canal. During the last year two patients in this condition were seen in the clinic, both with cerebrospinal fluid at times leaking into the large bowel. They had suffered from many attacks of meningitis, and

they were both brought in during attacks, and died. It was then learned that their suffering had been caused by this very unusual condition.

The invertebrate was unfortunate in being controlled at the site of intake of food; his mouth was completely surrounded by the nervous system, a ring of it giving touch and taste. With increased development of the nervous system, the olfactory nerves with the brain areas for muscle function, the ganglions for sight and later for hearing, were placed over the cephalic stomach. The digestive fluids developed from areas of cells on each side of the stomach, small masses of cells which resemble the cells of the liver and pancreas. The stomach emptied into a single straight gut, the intestinal system, which accomplished little work by peristalsis, mostly by cilia just as in the trachea and in the fallopian tubes. It is said that at the third month of life of the human embryo the lower third to half of this tube in the center of the spinal cord, which represents the old straight gut of the invertebrate, is filled with loose hair-like cilia; later these are absorbed.

Kubie and Fulton recently reported two new cases of teratomatous cysts of the spinal cord and reviewed many from the literature. The cases are all most interesting, and corroborate the claims that the ciliated columnar cells in the tumors with mucus were remnants of the evolution of the straight gut with smooth muscle and similar cells of the invertebrate. Some of the cells and new-growths are found in the ependyma and choroid areas of the ventricles, the digestive fluid areas of the invertebrate stomach. The report is excellent, but stops just short of the true solution of the anomaly. The more brains the invertebrate developed the more difficult it was for him to get food into his stomach. Some of the invertebrates, however, became quite large; the ancient giant sea lobster was five feet long. The semi-mucous-membrane structure which we now call the ventricles of the brain and which has been enfolded by the enormous development of man's greater nervous system still retains on the sides of the ventricles in the ependyma of the choroid plexus the area which produces the cerebrospinal fluid; it is possible that some chemical stimulus acting on the secretory part of this structure could make fluid enough, by stimulating the cells which once made the digestive fluid and now make cerebrospinal fluid, to cause the development of hydrocephalus or hydrocephalus and spina bifida or spina bifida alone with less of the fluid formed.

I once saw a small child who was born with spina bifida which ruptured early. The skin was reddened and softened about it just as occurs in a leaking pancreatic cyst or fistula of the duodenum. In the early weeks of the life of the human embryo the spinal cord and the spinal column grow equally in length, and in the fourth month the spinal column rather rapidly outgrows the spinal cord. The nerves are brushed downward with this growth and the cauda equina develops. The legs have developed shortly before this, and nature, to prevent traction on the nerves to the legs, fused the outer covering of the spinal cord, the dura and pia, with the lower end of the central tube of the spinal cord and attached this onto the end of the coccyx. Thus as the spine grew it took traction off the nerves. But if this filament was not strong enough, if it stretched too much, or if it pulled off, then the child should be born with club feet, a condition occasionally seen with spina bifida. In this area, too, are lost-out particles of nervous system, which, in the embryonic stage, may, through some stimulus, develop growths, the nerve tissue tumors of the pelvis and those about the coccyx and sacrum. The most common dermoids or partial dermoids are the pilonidal cysts in which this terminal filament is connected near the coccyx close to the skin; it draws the skin in, making canals with a small bunch of hair projecting from them, or sometimes true closed dermoids.

Today it is possible to determine the presence of twins long before birth by the use of the stethoscope and the roentgen-ray. In the old days an excess of fluid or the large abdomen of the pregnant woman made her physician think of the possibility of a defective baby. Is there a change in the chemistry of the fluid to change embryonic develop-

ment, as found by Loeb, who in experimenting with frog's eggs found that by developing the fertilized egg in 0.5 or 0.6 per cent sodium chloride solution anomalies of the nervous system frequently resulted?

I have, of course, discussed only a few of the anomalies found in man. The others are equally interesting but they are not often found associated with exstrophy of the bladder, the subject of the evening's discussion.

THE ADRENAL FACTOR IN HYPERTHYROIDISM[1]

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ADRENALIN causes increased heart action and increased pulse pressure; hyperthyroidism causes the same.

Adrenalin causes dilatation of the vessels of the skin and sweating; hyperthyroidism causes the same.

Adrenalin causes dilatation of the pupils; hyperthyroidism causes the same.

Adrenalin increases metabolism; hyperthyroidism does the same.

Adrenalin tends to produce hyperglycæmia; hyperthyroidism does the same.

Adrenalin has a profound effect on the gastro-intestinal tract; hyperthyroidism has the same.

Adrenalin activates the nervous system; hyperthyroidism does the same.

The symptoms of hyperthyroidism, then, are the same as the symptoms of adrenalism. It would appear, therefore, that hyperthyroidism as it is revealed by its symptoms should more appropriately be called hyperadrenalism than hyperthyroidism. We shall presently see, however, that neither of these terms, hyperthyroidism or hyperadrenalism, is adequate to describe this disease.

The injection of adrenalin in a patient having hyperthyroidism produces an exaggeration of every symptom of hyperthyroidism. On the other hand, no amount of thyroid extract or of iodine can immediately cause any symptom of hyperthyroidism.

Experimental evidence also confirms the clinical observation that as the thyroid activity is increased, the effect of adrenalin on the organism is stepped up in a sort of mathematical ratio. One fact alone is sufficient to show that the production of adrenalism is dependent on the thyroid, namely, that in myxœdema the injection of adrenalin has little or no effect. In the presence of thyroid deficiency, adrenalin loses its power, but it is also true that in the presence of adrenal deficiency, as in Addison's disease, there can be no hyperthyroidism. Therefore, the thyroid and the adrenal glands are each equally essential to the production of hyperthyroidism.

Let us now consider the exciting causes of hyperthyroidism, especially the conditions that may cause thyroid crises. These data are significant, for they disclose that the chief, perhaps the only, causes of thyroid crises are those factors that cause an increased output of adrenalin. The factors which cause thyroid crises are the following: (a) pain; (b) emotional excitation; (c) foreign proteins—autointoxication, wound secretion, focal infections, infectious diseases; (d) asphyxia; (e) inhalation anæsthesia; (f) hæmorrhage; and (g) the injection of adrenalin.

These are the only factors known to me clinically that can precipitate a thyroid crisis. What common factor in asphyxia, hæmorrhage, physical injury, emotional strain, infection, etc., is responsible for the thyroid crisis? Obviously it is adrenalin, for each of these factors except the injection of adrenalin is capable of producing an increased output of adrenalin. Moreover, no other recognized clinical condition causes an increased output of adrenalin.

On the other hand, what factors do not cause an increased output of adrenalin and do not aggravate a case of hyperthyroidism? Neither food, nor drink, nor electrolytes, nor narcotics, nor stimulants, nor sleep, nor rest,

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nor a normal daily routine, nor any other factor in the whole external and internal environment can either cause an increased output of adrenalin or precipitate a thyroid crisis.

We now see that the expression of the disease is the result of increased adrenal activity but only in association with increased thyroid activity. I have seen no cases in which hyperthyroidism has been associated with a normal thyroid gland, and I have not seen a single case of hyperthyroidism in which the patient's condition did not improve after the removal of an adequate portion of the thyroid gland. The adrenal and the thyroid factors both play vital rôles in the production of hyperthyroidism. We shall presently see what is the rôle of each of these, but before taking up that point, we must also identify one other essential factor, namely, the nervous system.

The great rôle of the nervous system is shown by the following facts:

1. In hyperthyroidism, physiologic rest is one of the most effective forms of treatment.

2. In many cases the cause of the disease can be traced to excessive nervous strain.

3. Ligation of the superior thyroid artery, which produces a break in the innervation of the thyroid gland, profoundly modifies the disease and usually converts a hyperplastic gland into a resting gland.

To these should be added the following biologic facts:

1. That the disease occurs only in members of the human race, the principal characteristic of which is the development of the nervous system—especially of the brain; and

2. That among the human race hyperthyroidism rarely occurs in the inferior peoples nor in the stupid and criminal classes of the white race.

All these facts indicate that the nervous factor belongs to the picture almost as definitely and potently as do the adrenal and the thyroid factors.

It is clear, then, that there are three dominant factors in the production of hyperthyroidism—the thyroid gland, the adrenal glands, the nervous system. Let us now introduce experimental and clinical evidence indicating the rôle of each of these factors.

Let us recall that the brain is the master organ and that through its nerve connections it drives the organism; that oxidation is the source of that driving force; that the trillions of cells of which the organs are composed are electrochemical units; and that each of these cells is surrounded by a film, the variation in the permeability of which causes a variation in the activity of the cell. One would expect, therefore, to find some organ the sole function of which is that of changing the permeability, hence the activity of the cells of the organism. This organ is clearly the thyroid gland. The thyroid hormone causes a specific increase in the permeability of all the cells, hence an increased activity of the trillions of cells of the body. An important consequence of this specific action of the thyroid gland is an increase in the electric potential and in the electric conductivity of the tissues. Increased electric potential determines the range of the functional power of the organs and tissues. Comparable to an electric battery, a high potential means a high energy-producing power, hence high ability for work. In other words, the rôle of the thyroid is to build up the potential in the cells of the working organs and to increase the conductivity of the tissues. These important facts have been verified by measurements made in our Biophysics Laboratory in collaboration with A. F. Rowland and Maria Telkes.

Furthermore, our researches have shown that in myxœdema both the conductivity and the potential of the tissues are exceptionally low, a fact in harmony with the clinical observation of the stupor and low metabolism of myxœdematous patients — the opposite state to that in hyperthyroidism.

Apparently we have disclosed the definite rôle played by the thyroid gland. But we must at once point out a rôle which the thyroid gland cannot play. The thyroid cannot discharge the cells of the organs and tissues, the potential of which it builds up. This is inevitable, as in man-made batteries the charging and the discharging mechanisms are separate and distinct mechanisms. Obviously, the mechanism for charging a battery can no more discharge the battery than can the mechanism that discharges a battery charge it.

The animal cells, the tissues, the organs—the whole organism, in which the rate of accumulating a charge is governed by the thyroid gland, has no more power to discharge itself in work done than is possessed by a plant cell. Were there no discharge mechanism, a healthy human would be as quiescent as a healthy turnip. But the human has a marvelous discharge mechanism which is entirely separate from the thyroid gland—the discharging mechanism is the nerve-adrenal combination.

Our researches on electric potential have shown that in each case the effect of the injection of adrenalin, the effect of nerve stimulation, and the effect of electric stimulation is to discharge the potential as energy is drawn off to do work. The active work—excessive work in hyperthyroidism—is due to the action of the nerve-adrenal mechanism.

The thyroid mechanism is the charge-up mechanism; the adrenal-nerve mechanism, the discharge or work mechanism. Obviously, therefore, neither of these can substitute for any one of the others. Nor can any one work without the others.

How interesting it is, therefore, that the principle of anoci-association, which has been evolved on the basis of clinical experience in cases of hyperthyroidism, has as its objective the avoidance of these very factors—pain, emotion, infection, anæsthesia, hæmorrhage; and how naturally may one expect a high recovery rate after the removal of an excess portion of the charge-up mechanism—the thyroid—if stimulation of the discharge mechanism, the death-dealing mechanism—the adrenals—is avoided by the elimination or minimization of these factors. The recovery rate is indicated by the following statistics: Among 1244 cases in which information is available, 1219 or 97.9 per cent of the patients are reported to be in good or fair condition more than 1 year after operation.

Furthermore, how clear and logical is the postoperative care, which has as its chief objective as complete an avoidance of excitation of the discharge mechanism during the postoperative period as is attained during the operation. As stated, there is strong evidence that the activity of the thyroid is under the control of the discharge mechanism—namely, the nerve-adrenal mechanism.

Thus, to recapitulate:

1. Nervous excitation is known to be a cause of many cases of hyperthyroidism.

2. Relief from nervous strain leads to relief of symptoms and the return of the hyperplastic gland to the normal.

3. In cases of hyperthyroidism the division of the nerve supply of the thyroid causes great improvement, sometimes even a complete disappearance of symptoms, and the return of the hyperplastic gland to the normal.

4. Every one of the known excitants of hyperthyroidism, namely, infectious diseases, focal infections, emotional excitation, etc., involves nerve excitation which in turn produces an increased output of adrenalin. The adrenalin, in turn, has the power of activating the thyroid.

In view of the above considerations it would appear that a primary adrenalectomy would have both immediate and remote advantages in cases of extreme hyperthyroidism. The immediate advantages are indicated by a comparison of the early postoperative course of patients after thyroidectomy and after adrenalectomy. After thyroidectomy, the patient is at first extremely nervous and difficult to quiet; after adrenalectomy, the patient is usually quite calm and rests well. By adrenalectomy, therefore, the acute exacerbation of the hyperthyroidism, which is so dangerous in cases of extreme hyperthyroidism, is avoided. After thyroidectomy, the pulse rate is usually very rapid and remains so for several hours, often increasing in rate rather than decreasing; after adrenalectomy, the pulse rate gradually drops. More sedatives are required after thyroidectomy than after adrenalectomy. Excessive perspiration is noted after thyroidectomy and but a moderate amount after adrenalectomy. As for the remote results, the permanent lessening of the discharge mechanism, that is, of the adrenal tissue, lessens the probability of recurrence of the disease after the removal of a portion of the hyperactive thyroid.

It is obvious that we are now approaching an understanding of hyperthyroidism and are still increasing our ability to cope with

the disease successfully. The foregoing considerations at least supply an interpretation of the exciting causes of the symptoms and the clinical course of the disease. They offer an interpretation of the dominance of the brain in definite physical terms; they point out clearly the physical basis for psychic management and for the rôle of focal infections and infectious diseases; they show that the thyroid, the adrenals, and the nervous system are each affected by each of the others and that, in turn, each affects each of the others—a necessary arrangement for the primitive energy-transforming system—a system which transforms potential into kinetic energy.

The nerve receptors are the means whereby this energy system adjusts the organism to the environment, etc. The nervous system is passive until activated; the adrenal is quiescent until activated; the thyroid is quiescent until activated. In the rôle of the automaton thus created, the thyroid is driven to govern the potential and the permeability and with it the activity of the countless cells of the organism. The adrenal and the nerve mechanism cause a discharge of energy which is manifested by work done—emotion, exertion, etc.

When one considers this correlation of the thyroid, the adrenals, and the nervous system as evidenced by clinical observation and by experimental data, especially by the evidence accumulated in biophysical researches, it becomes clear that a new name must be given to the disease which we have formerly associated only with the thyroid by the term hyperthyroidism.

TABLE 1.—SUMMARY

Mortality last 1000 thyroidectomies		0.6 per cent*
Total operations on the thyroid		18,331
Total thyroidectomies	13,090	
Total ligations	5,241	
Total thyroidectomies for hyperthyroidism		9,463
Adrenalectomies for hyperthyroidism		10
Unbroken series since July 23, 1928		
Thyroidectomies and lobectomies		426
Ligations		94
Grand total		520

*This includes one partial operation for malignant thyroid with metastasis to the lung.

PARAVERTEBRAL ANÆSTHESIA IN UROLOGY

With a Report of Its Use in One Thousand Cases of the Kidney and Ureter[1]

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AND
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SINCE the disadvantages that may be encountered in the use of general anæsthesia are well known, a detailed discussion of them is hardly necessary. The heart, the lungs, and primarily the kidneys are not indifferent in their action when ether, chloroform, or nitrous oxide is used as an anæsthetic. The influence of these anæsthetics on kidney function has long been an object of study. Many authors (Thompson, Haines, and Milliken) have been able to demonstrate that there is delay during general anæsthesia in the appearance of indigo-carmine from both kidneys.

Grondahl examined the urine of 75 patients after ether narcosis and was able in 36 per cent of the cases to show albumin.

S. Pascual's work on the effect of general anæsthesia on kidney tissue is of interest.

In normal structure, the epithelial cells in the convoluted tubules have a striated border which is thought to play an important part in urinary secretion. Pascual made histological examination of kidney tissue after a general anæsthesia had been used and found that this striated border of the epithelial cell was lacking. The general result of narcosis is an oliguria without a disturbance in the concentrating power of the kidney. However, a patient whose kidneys have a lowered concentrating power will develop a postoperative nitrogen retention, if he receives a general anæsthetic. If we lay aside the disadvantages of general anæsthetics, we still find that in various pathological conditions, paravertebral anæsthesia is undoubtedly the method of choice. In arteriosclerosis, severe cardiac lesions, myocardial degeneration, tuberculosis of the lungs, and bronchial asthma, the use of paravertebral anæsthesia has a distinct advantage.

Local anæsthesia has come to assume an important rôle in modern urological surgery. Frequently the surgeon is confronted with cases in which the use of a general anæsthetic is contra-indicated. If a renal deficiency exists before operation or the remaining kidney demands operative attention, then local anæsthesia becomes the method of necessity rather than of choice.

Certain conditions must be fulfilled in order to insure a successful result with paravertebral anæsthesia: The surgeon must have (1) the co-operation of the patient; (2) he must use a good anæsthetic; and (3) he must use the proper technique.

THE PATIENT

In working with any form of local anæsthesia, it is of paramount importance to gain the confidence of the patient and make him less apprehensive. In this Clinic, all of the operations are performed under local anæsthesia, and the patients have come to expect this. As a result the patient comes to operation with a feeling of security and confidence. Persons of low mentality who are of a distrustful frame of mind and cannot overcome this tendency (neurasthenic or hyperæsthetic individuals) are not proper subjects for paravertebral anæsthesia; hence another form of anæsthesia is advisable in such cases.

The evening before operation the patient is given 0.5 gram of veronal. Thirty minutes before the patient is taken to the operating room, 0.02 gram of morphine sulphate are injected. The veronal insures a night's sleep, the morphine lowers the sensibilities.

ANÆSTHETIC

Novocain is used in this Clinic as the anæsthetic of choice. It has proved to be the least toxic, most reliable, and least expensive anæsthetic. The novocain solution is prepared from novocain-adrenalin tablets, which are dissolved in a physiological salt solution.

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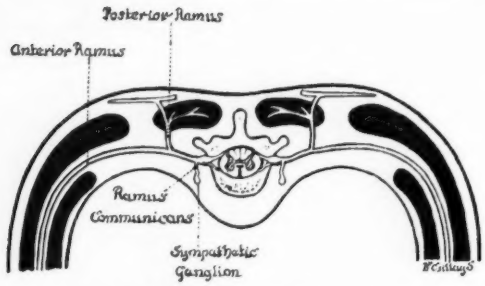

Fig. 1. Sagittal section showing the course of an intercostal nerve (after Corning).

Each tablet contains 0.125 gram of novocain and 0.000125 gram of adrenalin. The 1 per cent solution of novocain, which is the strength used to produce paravertebral anæsthesia, is prepared by dissolving 8 of these tablets (pharmaceutical division of Bayer-Meister Lucius) in 100 cubic centimeters of physiological salt solution. This solution must be freshly prepared before each operation and sterilized by boiling. It is then cooled and ready for use. The adrenalin content of the anæsthetic, by its action in constricting the blood vessels, makes possible a slow absorption of the novocain. Thus, the action of the novocain is more intensive and lasting, being effective for a period of 2 hours.

TECHNIQUE

In discussing the technique necessary for a proper administration of the anæsthetic, it may not be amiss to review a few anatomical relationships, primarily the course of the nerves to be anæsthetized. The region of the abdominal wall that must be incised in operations upon the kidney and ureter is innervated by the intercostal and lumbar nerves. The intercostal nerve, as it emerges from the intervertebral foramen, travels with the artery and vein of the same name, in a sulcus, on the lower and inner border of the rib. These structures, which lie behind the pleura, proceed with the ribs anteriorly. Where the intercostal nerve makes its exit from the intervertebral foramen, it gives off a communicating branch, the ramus communicans, to the sympathetic chain. This sympathetic chain, which is connected with the intercostal nerve

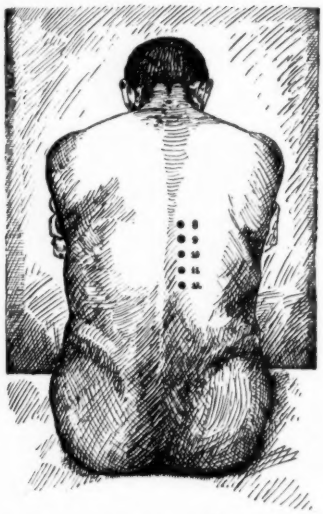

Fig. 2. The position of the patient during the carrying out of the paravertebral anæsthesia. Points from *8* to *12* indicate intracutaneous wheals made with novocain solution to designate the sites of injection.

through the rami communicantes, runs down on each side of the vertebral column. It is composed of chains of ganglia, each ganglion lying in an intercostal space. The intercostal and lumbar nerves, together with the nerve arising from the twelfth thoracic (iliohypogastric nerve) and the nerve arising from the first lumbar (ilio-inguinal), innervate the abdominal wall. The sympathetic fibers innervate the kidney, the adrenal, the ureter, peritoneum, and the other abdominal organs. It is possible, therefore, by directly infiltrating the intercostal and lumbar nerves at the point where they emerge from the intervertebral foramen, which is the site where the rami communicantes takes origin, to produce an anæsthesia, not only in the abdominal wall, but especially in the kidney and ureter.

In order to obtain a good anæsthesia, in operations of the kidney and ureter, we block from the eighth to the twelfth dorsal nerves, that is, the last 5 intercostal nerves and the first lumbar nerve. If it becomes necessary to work on the lower ureter, it is more advantageous to anæsthetize in addition the second and third lumbar nerves. Recently, some urologists have advocated blocking only

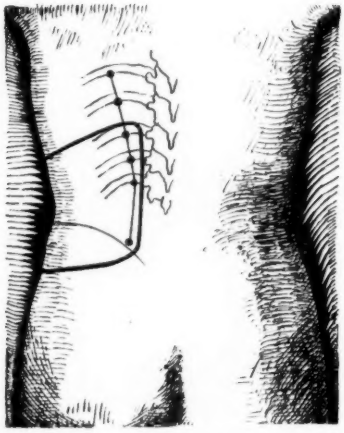

Fig. 3. Points of injection over the lower border of ribs (eighth to twelfth) and the iliac crest. The space marked out by the dark lines shows the area that is anæsthetized through both the paravertebral anæsthesia and the local infiltration (after Braun).

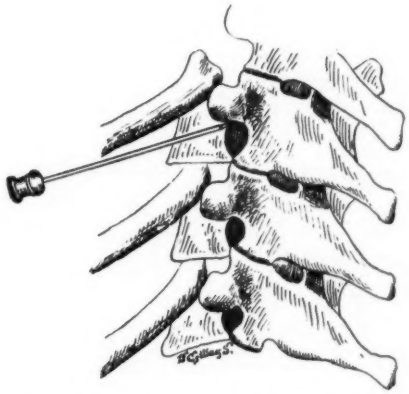

Fig. 4. The needle is shown being directed parallel with the rib to the transverse process of the vertebra and the intervertebral foramen.

the eleventh and twelfth dorsal and first lumbar nerves, while others have attempted to work with an anæsthesia of only the twelfth dorsal and first lumbar nerves (von Lichtenberg).

The technique of paravertebral anæsthesia, as used in this Clinic, is as follows: the patient sits upon the operating table with his legs hanging over the edge. He is told to cross his arms in front of him, bend slightly forward, and arch his back. Aided by an assistant he maintains this position. In this way the intercostal space is increased and the ribs can be more easily palpated. The lower angle of the scapula, which marks the location of the seventh rib, is located. The next lower rib is the eighth. At this point we begin the anæsthesia. In stout patients it may be difficult to make out the lower angle of the scapula. If the patient is told to move his arm and shoulder up and down, the scapula will move also and in this way give us a landmark.

At the level of the eighth rib we palpate the spinous process of the vertebra. Two to two and a half fingers' breadth lateral from this point, and on a level with the lower border of the eighth rib, we place a small wheal of novocain in the skin, as a guide. This is done in the same manner with each rib until the twelfth is reached. Then a wheal is made over the iliac crest just lateral to the long muscles of the back. Using a thin needle, from 9 to 12 centimeters in length, we begin at the eighth rib where the first wheal was made. The needle goes through the skin and underlying soft structures until the lower border of the rib is reached. There 3 or 4 cubic centimeters of 1 per cent novocain solution are injected. Care must be taken not to injure the pleura: In stout persons, in whom the needle must pass through a thick layer of soft underlying tissue, it may be difficult to find the lower border of the rib. In such cases, one can carefully use the needle directly as a guide to the body of the rib, taking care, however, not to injure the sharp point of the needle, which would hinder the sense of feeling necessary for the fine work to follow. When the rib is found, the needle is moved slowly downward until the lower border is reached. By this method, it is possible in very stout patients to inject the novocain precisely under the lower border of the rib and thus obtain a good anæsthesia. Contrary to the view held by other workers, who state that it is immaterial whether the upper or lower border of the rib is injected, we have found that in view of the small amount of anæsthetic introduced, the best result is obtained when the injection is made exactly at the lower border.

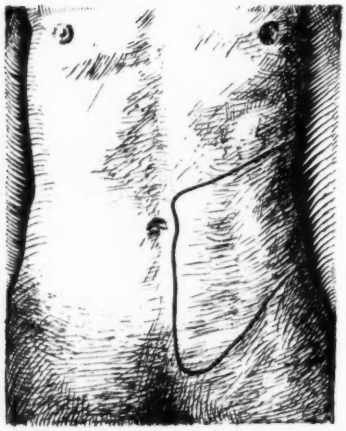

Fig. 5. The lines mark out the anterior portion of the abdominal wall that is infiltrated locally in operations on the kidney and ureter (after Braun).

It should be stated that in the anæsthetizing of the intercostal nerve, the so-called intraneural injection is made difficult on account of the situation of the nerve in the costal sulcus. Therefore, a perineural infiltration must be given. Three to four cubic centimeters of the novocain solution are sufficient to reach and anæsthetize the intercostal nerve. When the novocain has been injected against the lower border of the rib, the needle is withdrawn a few centimeters and its point is directed toward the vertebra and parallel with the rib. The needle is slowly carried forward until a bony resistance is felt, which is the vertebra. We inject 3 or 4 cubic centimeters of the novocain solution at this point. This technique is carried out from the eighth to twelfth rib.

The injection at the rib border blocks the intercostal nerve, while the injection of novocain at the vertebra anæsthetizes the rami communicantes and through this there results an anæsthesia of each segment.

From the last wheal on the twelfth rib and then that over the iliac crest, the infiltration is carried downward in a fan shaped manner using from 20 to 25 cubic centimeters of novocain solution. This results in a blocking of the ilio-inguinal nerve.

In operations on the lower ureter, the lumbar nerves are anæsthetized in the following manner. The lumbar nerves, as they emerge from the vertebral foramen, take a direct and straight path downward. To block these nerves, we palpate the spinous process of the lumbar vertebra and 3 centimeters lateral from this point we introduce a needle going through skin and muscle in the direction of the transverse process of the lumbar vertebra. When the needle meets bony resistance, 3 to 4 cubic centimeters of the solution are injected. This is carried out with the first, second, and third lumbar vertebra.

After the regional anæsthesia is completed, the line of incision is infiltrated by means of the usual method, first subcutaneously and then deeper into the muscle layers.

In operations on the kidney, 150 cubic centimeters of novocain solution are used. In cases in which the ureter is laid free in its entire length or one meets with a very thick abdominal wall, an additional 50 cubic centimeters of the solution must be used. This is for the anæsthesia of the lumbar nerves and to provide for the longer incision.

At this Clinic the results with the technique for paravertebral anæsthesia described have been practically 100 per cent successful in providing good anæsthesia.

It happens not infrequently that when the renal pedicle is clamped, the patient experiences some degree of pain. However, in most cases this is negligible. Finsterer and Torek have described a method of paravertebral anæsthesia in which they recommend, after isolation of the kidney, the injection of a small amount of novocain into the pedicle and surrounding peritoneum. Other authors advocate that a light ether anæsthesia be given during the ligation of the renal artery and vein. It has not been found necessary to apply either of these methods in the work at this Clinic.

Some workers, who perhaps have not had the opportunity to use paravertebral anæsthesia in a large number of cases, are of the opinion that it is not practical for general use in urological surgery. Von d. Huetten, on the basis of 7 cases, reports that this method of anæsthesia is complicated and difficult. In our review of 1,000 cases with paravertebral anæsthesia, we are able to state that the method

is simple and uncomplicated. A little practice enables one to give this form of anæsthesia with assuredness.

It has been said, in speaking of the disadvantages of paravertebral anæsthesia, that its administration requires time. In a clinic with a full operating schedule, too much time would be necessary to carry out this anæsthesia. However, this is not a real objection since, with a good technique, it can be given in about 10 minutes. An assistant, having scrubbed earlier, can complete the anæsthesia by the time the operator is ready to begin work. When one operation must follow another, in order to avoid delay, an assistant leaves the table a short time before the operation is finished and prepares the next case.

The toxic action that results in the use of an amount of novocain solution necessary for this anæsthesia is said to be a disadvantage and danger. We can state on the basis of 1,000 cases, in some of which 2 or 3 grams of novocain were given in ½ to 1 per cent solution, that we have not seen any toxic action from the drug. Furthermore, no untoward results have occurred from the adrenalin content of the solution. In nervous patients, the drug may at times give rise to slight symptoms, such as cardiac palpitation, a rapid and small pulse, dizziness, cold sweats, or vomiting. Collapse has not occurred in a single case. The slight symptoms just mentioned are ascribed to cerebral effects due to the absorption of novocain. It is questionable if the morphine given pre-operatively is not responsible in part for these symptoms.

Finsterer describes another disadvantage of paravertebral anæsthesia that makes its use impractical as a routine procedure. He has observed in a few instances that the dura can protrude into the intervertebral foramen and even bulge out to the point where the sympathetic ganglia is situated. In view of this, it is possible, by injecting according to the technique of paravertebral anæsthesia, for the needle to enter the dura. Thus, a large amount of novocain entering the dura could produce serious effects. Kappis, in reviewing 32 kidney operations under paravertebral anæsthesia, reports 1 death. In this case, he was able to demonstrate the presence of novocain in the spinal fluid. Among the 1,000 cases reported in this article, no similar experience has occurred.

It has also been said against this form of anæsthesia that the adrenalin in the solution gives rise to a secondary dilatation of the blood vessels and thus makes liable a postoperative hæmorrhage. Careful hæmostasis will avoid this. Here we can state that in our series of cases there has been no postoperative hæmorrhage that could be ascribed to the action of the adrenalin. Druener recommends that in order to avoid the injection of the solution into the blood vessels, the infiltration be carried out from layer to layer beginning with the operative wound. Kappis first infiltrates his line of incision, cuts down, uses a splanchnic anæsthesia, and then infiltrates the adipose capsule of the kidney. He claims that by thus injecting in two stages he avoids the danger of introducing adrenalin into the larger vessels.

Splanchnic anæsthesia is recommended by some authors. The technique necessary for this method is more complicated and dangerous than is the paravertebral method. Investigations show that splanchnic anæsthesia influences kidney function.

A. Schmidt and P. Siwan found that several hours after splanchnic anæsthesia there was a sharp decrease in the urinary output. They observed this in one-third of their cases. In 25 per cent of the cases there was a decrease in the output of nitrogenous products in the urine. Salt excretion was practically uninfluenced.

The administration of splanchnic anæsthesia is not without danger. Kappis, in his technique, introduces a needle at the lower border of the twelfth rib posteriorly. The needle is directed upward and medial for a distance of 7 centimeters, which is the point where the semilunar ganglion lies, and where the splanchnic major and minor nerves take origin. The novocain solution is injected at this place. It must be remembered that on the right side, the vena cava and on the left side, the aorta are close to the semilunar ganglion. Therefore, in carrying out a splanchnic anæsthesia, the danger of injury to these great vessels is not remote.

TABLE I—SUMMARY OF CASES

Indications for operation	Type of operation	Case
Tuberculosis of the kidney	Nephrectomy	508
Kidney calculi	Nephrectomy	87
	Nephrotomy	26
	Pyelotomy	73
Ureteral calculi	Ureterotomy	43
Hydronephrosis	Nephrectomy	70
Pyonephrosis	Nephrectomy	49
Pyelonephritis and nephritis apostematosa (surgical kidney)	Nephrectomy	32
	Decapsulation	14
Kidney tumors	Nephrectomy	32
Movable kidney	Nephropexy	15
Hæmorrhagic nephritis	Decapsulation	12
Tumors of renal pelvis	Nephrectomy	6
Polycystic kidney	Exploratory	4
	Nephrostomy	3
	Nephrectomy	2
Ureterovaginal fistula	Nephrectomy	
Congenital anomalies of the kidney:		
a. Aplasia	Exploratory	
b. Hypoplasia	Nephrectomy	6
c. Horseshoe kidney and calculi	Nephrotomy	13
		2
d. Double kidney	Nephrectomy	6
	Heminephrectomy	2
Large solitary serous cyst	Excision	3
Echinococcus cyst kidney	Excision	1
TOTAL 1,000		

Many surgeons prefer and recommend the use of spinal rather than paravertebral anæsthesia, because it is simple and requires only one site of injection. In view of the serious results that are often seen following spinal anæsthesia, we believe that in kidney surgery, paravertebral anæsthesia offers a safer and better method.

Rannuci, in a series of 67 cases, studied the effects of spinal anæsthesia on kidney function. He found that, following such anæsthesia, the urinary output is diminished and the urea nitrogen of the blood is increased. In one-third of the cases, he found albumin in the urine. This he explains as being due to the action of the novocain on the cerebral centers. Conducting his investigations along the same lines in cases in which paravertebral anæsthesia was used, he was not able to find any changes in the blood, or urine, such as were found in the spinal anæsthesia cases.

In all operations that they carried out under spinal anæsthesia, Abadie, Baldous, and Dornier noticed that there was an increase in nitrogen of the blood. E. Bamberger reports his observations in 166 cases operated upon under spinal anæsthesia. He found regularly an increase in body temperature. This occurred between the fifth and sixth days postoperative; in other cases, between the eighth and twelfth days; or sometimes this fever developed at both these periods. With the increased temperature, meningeal symptoms and also severe headaches appeared. There was an increase in the nitrogen products of the blood and a decrease in the output of phenolsulphonephthalein and indigocarmine.

In addition to these slight and transitory symptoms which spinal anæsthesia produces, some authors report more harmful results. Salleras observed a case in which, after an operation under spinal anæsthesia, a paralysis of the detrusor muscle of the bladder occurred. This patient developed incontinence of the urine and fæces which persisted for 3 years. Salleras stated that this paralysis was due to an injury of the urinary and defæcation medullary centers by the needle.

The influence of paravertebral anæsthesia on the kidney function was studied by K. Lion, who observed 200 cases. He found in his series that there was no increase in the urinary output or urine salts after paravertebral anæsthesia.

Protopapow, Neuwirth, and Andler do not confirm the results of Lion. They are able to report that with this anæsthesia no evidence of disturbance in kidney function has been observed.

In writing of his experiences with paravertebral anæsthesia, Lowsley reports that the blood pressure is not lowered as in spinal anæsthesia, nor is it increased, as frequently occurs with inhalation anæsthesia. He further states that the danger of lung complications is practically negligible. He sees as a great advantage the fact that the patient can partake of liquids immediately after the operation, which is of prime importance in kidney surgery.

If we review the various methods of narcosis described in this article and compare the disadvantages of inhalation, splanchnic, or spinal anæsthesia, with the advantages to be gained by using paravertebral anæsthesia, we cannot help but feel that in the surgery of the kidney and ureter, paravertebral anæsthesia is the method of choice.

In this Clinic, during the past 10 years, 1,000 operations on the kidney and ureter were performed under paravertebral anæsthesia. A summary of these operations is set forth in Table I.

The operations were all carried out in a most favorable manner. There were 10 cases in which a minimum amount of ether was given during the ligation of the renal pedicle.

In conclusion, we wish to state that in this Clinic most satisfactory results have been achieved with paravertebral anæsthesia.

SUMMARY

1. One thousand operations upon the kidney and ureter with paravertebral anæsthesia are reported.

2. The disadvantages, transitory or permanent, which arise with inhalation, splanchnic, or spinal anæsthesia, are not met with in the use of paravertebral anæsthesia.

3. A markedly neurotic and apprehensive patient should not be selected for this form of anæsthesia.

4. There is no contra-indication to the use of paravertebral anæsthesia, such as a low or high blood pressure, poor renal function, cardiac lesions, or pulmonary involvement.

5. The safety of this anæsthesia has been in our experience, absolute.

6. Its technique is uncomplicated and easily carried out.

7. It is the anæsthesia of choice in surgery of the kidney and ureter.

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PERIURETHRAL PHLEGMON (URINARY EXTRAVASATION)

A Study of One Hundred and Thirty-Five Cases[1]

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SO-CALLED "urinary extravasation" is an extensive fulminating phlegmon originating in or about the urethra and is usually accompanied by massive genital and perigenital gangrene. It claims a mortality of over 40 per cent. The lesion is always an infection, most often follows pre-existing urethral disease—notably stricture—but may be secondary to trauma. In private practice the condition is rarely encountered; in large hospitals it is frequently seen and, since convalescense is protracted, at Bellevue Hospital we are seldom without at least one such case in the wards of the Urological Service. As a rule, the diagnosis is correctly made and if the lesion has been observed once, one could scarcely fail to recognize it again. Treatment is surgical and should include immediate establishment of free urinary drainage together with inordinately wide incision of the involved areas.

During the past 14 years 135 patients with periurethral phlegmon have been admitted to Bellevue Hospital and all but 2 of these were treated on the Urological Service. Two infants, 3 and 6 weeks of age, were admitted to the Children's Surgical Service. This series constitutes the basis of this study.

ETIOLOGY

For anatomical reasons periurethral phlegmon is a disease peculiar to males. Although a third of our patients were between the ages of 45 and 60, no age was exempt (Table I). Two were infants without demonstrable urethral obstruction yet extensive operation was required. However, urethræ scarred and ulcerated by infections past and present, often weakened and dilated by the long continued urinary back-pressure secondary to stricture, offered the least resistance to infectious flare-ups. As a rule the inflammatory process is most severe at the site of stricture or old periurethral infiltration. In only 7 cases was an antecedent gonorrhœal infection denied. Twenty patients had been operated upon for stricture previously and 1 had been previously operated upon for extravasation.

Unless it follows recent trauma, the lesion is always of a primary infectious etiology. The "straddle injuries" of the perineum are the common trauma as in 4 of our patients. Following transverse rupture of the urethra by a crushing blow, urinary infiltration with accompanying infection and cellulitis ensues. Phlegmon may complicate fracture of the pelvis. We have recently observed a patient in whom extravasation followed urethral laceration by instrumental trauma. In Barwell's case (2) the lesion followed rupture of the membranous urethra during intercourse. While urethral obstruction (stricture) is found in the majority of extravasation cases (85 per cent in this series), its presence is not essential. Primary obstruction by urethral calculi has been observed by some, but such stones were not identified with our cases. In one patient, however, 11 calculi were found impacted behind a tight stricture at the junction of the pendulous and bulbous urethra. Suppurative periurethritis, adenitis (littritis, Cowperitis), and periadenitis with secondary localized urethral necrosis and phlegmonous infiltration account for certain cases without demonstrable stricture. There is still another group presenting an apparently intact urethra with no evidence of actual urinary infiltration yet characterized by massive gangrenous phlegmon and clinically indistinguishable from the lesions in which urinous infiltration is present. Some have designated these as idiopathic, but we believe them to be likewise of periurethral infectious origin. We observed 22 cases of the 2 latter types; 11 patients died. Because of the severity of the inflammatory reaction in most instances, it is quite impossible to

[1] From the Urological Service of Bellevue Hospital.

estimate to what degree the urethra has been involved by the periurethral process.

We have not considered as belonging to this non-stricture group a series of 295 cases of localized periurethral abscess observed during the period covered by this study nor cases of streptococcus scrotal and penile gangrene. Most periurethral abscesses are secondary to gonococcus infection, although many times other organisms can be demonstrated. While these abscesses are acute suppurative lesions pathologically not unlike the group designated as extravasation and are restrained by the same fascial planes as extravasation, we do not associate them clinically with the more extensive gangrenous phlegmons because they are localized. However, it is conceivable that by marked extension a previously localized periurethral abscess might become clinical extravasation, but the 2 cases of this series in which gonococci were demonstrated were both associated with stricture. Furthermore, we have not included those cases of urinary infiltration due to rupture of the bladder, ureter, or kidney.

Bacteriologically, streptococci, staphylococci, bacillus coli, bacillus perfringens, and various anaerobes are most often etiologically associated with extravasation. Certain observers, particularly the French, have emphasized the etiological importance of these anaerobic bacteria and have pointed out that without anaerobic invasion there will be no gangrene (5). However, a mixed infection is always present, the colon bacilli in many cases accounting for the nauseous stench emitted by the lesions.

ANATOMY

The extension of periurethral phlegmon is guided by certain anatomical structures, the external and internal pelvic fasciæ. A correct knowledge of the surgical anatomy of these planes is essential not only for a proper understanding of extravasation but also for its correct treatment. The present anatomical conception of these structures is indicated diagrammatically in Figure 1. The triangular ligament is the dividing line. It is a densely fibrous wall formed by two fascial layers, a firm anterior and thin posterior layer; it stretches across the pubic arch; and is attached anteriorly to the symphysis pubis, laterally to the ischiopubic rami, and posteriorly offers insertion for the all important Colles' fascia. Between the layers of the triangular ligament courses the penile vascular and nerve supply, and within its confines are the membranous urethra and the ducts of Cowper's glands.

TABLE I.—AGES OF PATIENTS

Years	Cases*
19 and under	2
20 to 29	12
30 to 39	25
40 to 49	28
50 to 59	39
60 to 69	26
70 and over	3
Total	135

*Infants 3 and 6 weeks old.

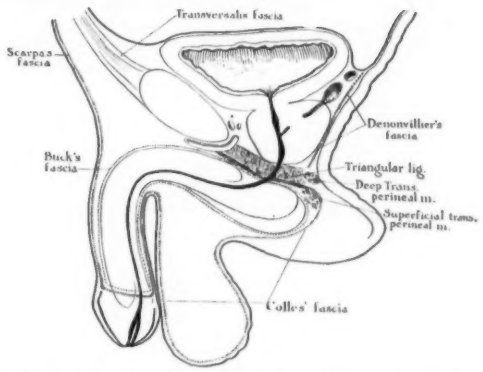

Fig. 1. Fasciæ of importance in urinary extravasation (after Wesson). Colles' fascia is concerned in nearly all extravasations and its abdominal continuation, Scarpa's fascia, is less frequently involved. Intrapelvic extravasations are least commonly observed.

As indicated, Colles' fascia, or more correctly the superficial perineal fascia, is firmly attached to the posterior border of the triangular ligament from which point it sweeps first backward and downward separating the deep and superficial transverse perineal muscles, then forward. It is firmly attached laterally to the ischiopubic rami, continues anteriorly under the perineal, scrotal, and penile skin to fuse with the deep penile fascia at the root of the organ. A compartment is thus formed—the anterior perineal triangular space—closed everywhere except at the base of the penis. Through this unprotected area extravasation

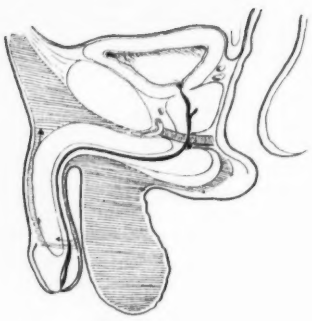

Fig. 2.

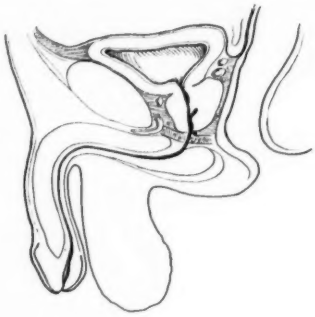

Fig. 3.

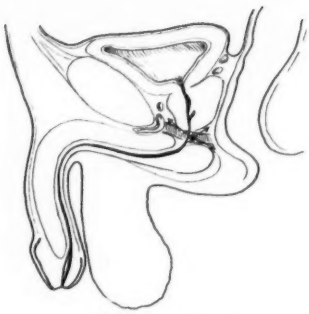

Fig. 4.

Fig. 2. Spread of extravasation when the primary focal lesion is anterior to the triangular ligament. A course bounded by Colles' and Scarpa's fasciæ is pursued.

Fig. 3. When extravasation originates on the pelvic side of the triangular ligament, retroprostatic, perirectal, and ischiorectal infiltrations are most commonly observed although perivesical and prevesical involvement may occur. Rupture into the peritoneal cavity occurred in 2 of our cases.

Fig. 4. If the extravasation originates within the triangular ligament, extension occurs in either direction, most often externally.

extends upward over the abdomen beneath Scarpa's fascia, which is the abdominal continuation of Colles' fascia. In the groin the firm attachment of the superficial abdominal fascia (Scarpa's) to Poupart's ligament quite generally prevents invasion of the anterior thigh. In only 2 cases did we find involvement in this latter location, both of these patients presenting extensive abdominal and genital infiltrations which had broken through the barrier at Poupart's ligament. The anatomical identity of the deep penile fascia of Buck and the fascia of Colles' is disputed, but Wesson (7), on the basis of injection experiments, has recently described these as separate structures (Fig. 1).

These are the main anatomical considerations and infiltrations which originate anterior to the triangular ligament will follow a course limited in the perineum and genitals by Colles' fascia and over the abdomen by Scarpa's fascia (Fig. 2). Perineal and scrotal involvement is noted first and, if neglected or improperly treated, penile, lower abdominal, and groin cellulitis immediately succeeds. Less often is the site of origin in the pendulous urethra, and here penile involvement may be localized by Buck's fascia or may be followed by upward extension over the abdomen. Rarely is the scrotum or perineum secondarily involved when the process originates in the pendulous urethra. If it originates on pelvic side of triangular ligament, retroprostatic extension most often occurs (Fig. 3). This usually invades the ischiorectal fossæ and may include the buttocks and inner upper aspects of the thighs. In one case coming to autopsy, retroperitoneal infiltration extended up the lumbar gutters to the kidney level. When intrapelvic infiltration points anterior to the urethra a prevesical or perivesical phlegmon results. At autopsy 2 cases showed this lesion with fatal secondary peritonitis. Unless pointing immediately into the ischiorectal fossæ, intrapelvic extravasation rarely manifests itself early enough to offer great hope of surgical cure.

When the primary inflammatory lesion is between the layers of the triangular ligament, the infiltration may point either way—toward the pelvis or, more often, externally—always taking the course of least resistance (Fig. 4). Occasionally, direct extension to the ischiorectal spaces may occur as we observed 4 times or may secondarily involve these spaces after penetrating Colles' fascia. The relative frequency with which the various structures were involved in our cases is indicated in Table II. The extent of the lesion depends upon the site of origin, the virulence of the infection, and the duration of the disease. We have seen cases of 72 hours' duration with infiltration extending from the perineum to the costal margins, and by this time genital gangrene had occurred.

PATHOLOGY

Most frequently there is urethral necrosis with subsequent perforation at or proximal to a stricture. This urethral gangrene may result from mechanical urinary pressure against a dilated, weakened, and infected membrane but is probably more often the result of acute local infection (flare-up) at the site of stricture or in an area of periurethritis. Mechanical factors explain some cases while infection explains all. In proof of this, patients have been observed in whom the origin of the infiltration was distal to a stricture. Afforded egress through the diseased urethra, the infected urine incites the widespread phlegmon. It has been proved by injection that sterile urine will not incite this process (8).

French observers (1, 2) in particular hold that actual urinary infiltration does not exist, that periurethral phlegmon is caused by bacterial invasion only—notably anaerobic, and that fluid in the tissues simulating urine is an acute inflammatory exudate. On the other hand, Kidd (6) repeatedly found a 2 per cent urea content in fluid obtained from the tissues at time of operation. In 2 cases at Bellevue in which this test was made, urea was found. Therefore, there may be phlegmon with or without urinary extravasation.

At onset the lesion is a cellulitis with associated extensive vascular thrombosis resulting in early gangrene. Invasion of the tunica vaginalis, spermatic cord, or corpora cavernosa or spongiosum seldom occurs although we have observed the incidence of each.

With chronic urethral obstruction, upper urinary tract dilatation and infection coexist and are dependent upon the duration and degree of the blockage. In 2 cases, no phenolsulphonephthalein was obtained in 2 hours' observation and in 3 others only a trace was found (Table III). Acute superimposed pyelonephritis is often encountered and in some instances is the immediate cause of death (10 cases).

TABLE II.—STRUCTURES INVOLVED BY EXTRAVASATION

	Cases
Perineum	94
Scrotum	127
Penis	60
Groin	26
Abdomen	34
Buttocks	4
Thigh	2
Gangrene present	77

TABLE III.—LABORATORY FINDINGS*

Phenolsulphonephthalein (2 hour return)	Cases
None	2
Trace	3
5 to 10%	5
11 to 20	7
21 to 30	9
40 to 50	6
Over 50	5

Culture	Cases
Gonococcus	2
Streptococcus	12
Staphylococcus	6
Bacillus coli	4
Gas bacilli (anaerobes not bacillus welchii)	3

Non-protein-nitrogen (mgm. per 100 c.cm. blood)	Cases
Under 35	27
36 to 50	19
51 to 75	7
Over 75	6
Highest	228

Creatinin (mgm. per 100 c.cm. blood)	Cases
Under 1	2
1 to 2	8
2.1 to 3	44
3.1 to 4	2
5	1

*Because periurethral phlegmon is always an emergency procedure, conditions seldom permit of pre-operative function tests, therefore nearly all of these records represent the first estimation after operation.

SYMPTOMS

The toxic symptoms of cellulitis overshadow all others save those of acute urinary retention when urethral obstruction is present. Marked local tenderness and swelling, chills and fever succeeded by toxic mental confusion often progressing to delirium and coma, are the outstanding symptoms. The intensity of both local and general symptoms is governed by the degree and duration of the phlegmon. Constitutional resistance is a feeble factor in the face of an overwhelming bacterial toxæmia such as that produced by the streptococcus, for example. The majority of these patients have long battled more or less fortuitously with stricture, periurethral abscesses, and renal infections. Little resistance indeed can these men offer to bacterial invasion with its attendant gangrene, more especially when the heart also has been damaged by cardiovascular degeneration.

Fig. 5. The characteristic appearance on admission. In this case penile gangrene was pronounced and scrotal gangrene was far advanced.

Fig. 6. Marked perineal and penile involvement. Although scrotal involvement is present in this case, it is not as advanced as is usually observed on admission to the hospital.

Dysuria and other urethral symptoms are usually pronounced. Urinary difficulty was noted by 72, diminished stream by 49, dribbling by 21, marked frequency by more than half, hæmaturia by 19, gleet by 18, and burning or painful urination by 35. Fourteen patients were admitted in acute complete retention and 8 were in chronic complete retention with overflow.

The onset of phlegmon is abrupt. Occasionally, patients have noted the presence of nodular periurethral infiltrations for some time previous to the acute onset. Most often the lesion is heralded only by acute dysuria immediately followed by swelling and the signs and symptoms of phlegmon. The duration of the phlegmon as noted by our patients is indicated in Table IV. Those lesions alleged to be of 2 to 3 months' duration (5) were obviously large periurethral infiltrations which had recently and suddenly burst their bounds.

TABLE IV.—DURATION OF PHLEGMON TO KNOWLEDGE OF PATIENT*

Days	Cases	Weeks	Cases
1	11	1	0
2	6	2	0
3	11	3	7
4	16	4	0
5	18	5	1
6	4	6	2
7	11	7	0
8–10	11	8–10	1
11–14	9	11–14	1

*Not stated 24 cases.

DIAGNOSIS

The correct diagnosis is often made by inspection. One finds a bulging perineum and a greatly swollen, purplish-red scrotum which looks as if it were about to burst (Figs. 5 and 6). The surface is frequently spotted with areas of greenish-black gangrene and emits the odor characteristic of decomposing flesh. A similar picture may be presented by the penis, groins, and suprapubic abdominal wall. The genitalia are often 6 to 8 times normal size. Palpation reveals this enlargement to be an œdematous cellulitis, and urethral instrumentation will usually disclose an obstruction. These patients look sick, they are septic, dehydrated, have rapid pulse and respirations, and may be delirious or even comatose. Nine of our patients were in coma when admitted to the hospital.

Extravasation must be differentiated from the massive œdema of cardionephropathy, cirrhosis, etc. In these latter cases, œdema

TABLE V.—ANÆSTHESIA

	Cases
General	73
Local	4
Spinal	54
None	3

Fig. 7. Postoperative appearance of lesion similar to that shown in Figure 5. The scrotal bisection and débridement are noteworthy as are also the freely swinging testicles.

elsewhere, particularly of the lower extremities, gives the diagnostic clew. Urinalysis will rule out diabetic gangrene although this is seldom an isolated lesion of the scrotum. We have recently seen a case of extravasation which had been operated upon under the mistaken diagnosis of strangulated hernia. There was present scrotal gangrene and universal cellulitis of the lower half of the abdomen. Herniotomy incision revealed advanced subcutaneous gangrene. A similar error was made some time ago in another case in which the extravasation first involved but one side of the scrotum.

Streptococcus scrotal and penile gangrene most closely simulates extravasation. There is lacking, however, a history of antecedent urethral disease, stricture cannot be demonstrated, perineal involvement is rare and always secondary to a genital inflammation, and in all cases studied by us (3) a pure culture of hæmolytic streptococcus longus was isolated. In most respects this lesion is not unlike an intense erysipelas of the genitals and incision fails to reveal evidence of periurethritis. Nevertheless, at Bellevue one case of this type was operated upon under the mistaken diagnosis of extravasation.

Hydrocele, orchitis, or acute epididymitis can hardly be confused with extravasation.

Fig. 8. Extensive incision, scrotal bisection, and perineal bladder drainage in an infant 6 weeks of age. An insufficiently incised œdematous penis is noted.

TREATMENT

The immediate establishment of free bladder drainage together with wide incision of the involved tissues and the administration of enormous quantities of fluids offers the only hope for these patients. Under spinal anæsthesia a perineal section is performed, the strictures are cut to admit the passage of a large sound to the bladder, and a perineal bladder tube is fastened in place. Inordinately wide drainage incisions are then made throughout the involved areas extending well into the margins of normal skin.

About half of our patients were operated upon under general anæsthesia (gas-oxygen-ether), but since 1920 we have been using spinal anæsthesia in most and since 1924 in all of these cases. Spinal anæsthesia is now the anæsthesia of our choice for most major urologic surgery (4). Four moribund patients were incised under local anæsthesia, 3 required no anæsthesia (TABLE V).

TABLE VI.—TYPE OF OPERATION PERFORMED—ALL WITH INCISION AND DRAINAGE OF SKIN

External urethrotomy	101
Internal urethrotomy	3*
External and internal urethrotomy	25
Incision and drainage only	4†
No operation	2‡
	134

*All died, insufficient drainage.
†No urethral obstruction found.
‡Moribund on admission, died before operation could be performed.

Fig. 9. Cutaneous repair is characteristically rapid and complete. *a* and *b*, 6 weeks after operation; *c* and *d*, 6 months after operation. The initial postoperative appearance was similar to that shown in Figure 7.

If the stricture is of filiform caliber, as in 18 of our cases, or is impassable, as in 12 others, the injection of a half ounce of methylene blue solution into the urethra will often aid in the recognition of the lumen of the canal when perineal section is performed (Table VI). With proper urethral surgical technique and an accurate knowledge of perineal anatomy one can usually contrive to enter the urethra, cut the strictures, and insert the perineal tube into the bladder. In those cases in which this is found impossible, suprapubic cystotomy must be done. Because new avenues of infection are thereby opened, particularly into the prevesical space of Retzius, one hesitates to perform cystotomy. More especially is this true when suprapubic cutaneous extension of the cellulitis has occurred. In no case of this series did the primary bladder drainage require suprapubic approach, although 5 patients in whom plastic urethral operations were later performed were subsequently drained from above. In those cases in which the location of the stricture was recorded, the sites were as follows: penile 12, bulbous urethra 22, bulbomembranous 15, and membranous 3.

Fig. 10. Fig. 11.

Fig. 10. Showing complete healing with testes anterior to the penis, often the result of cord shortening by induration. A small hydrocele is present on the right side.

Fig. 11. Thiersch grafts were used to accelerate cutaneous repair. This also shows the character and extent of the drainage incision for a moderate suprapubic extravasation.

Having established bladder drainage, one immediately incises the involved areas widely, bisecting the scrotum when necessary, and performing débridement of gangrenous portions. This frequently leaves the testicles swinging freely, but is life saving (Figs. 7 and 8). Incisions should be carried into the margins of healthy tissues. If this is not done, further infiltration may occur and necessitate re-operation for extension of incisions. We found that of 15 patients requiring re-operation because of further infiltration, 9 died.

"Timorous incision is the patient's death warrant" (Keyes), and not a few of our patients have been bilaterally incised from the perineum to the costal margins.

The preservation of free bladder drainage and an enormous fluid intake are chief of the postoperative measures. Upon renal function rests the battle for life in most instances. We not infrequently give at least 3 hypodermoclyses of 1,000 cubic centimeters each in 24 hours in association with a voluminous fluid intake by mouth and continuous rectal drip. If the heart continues to function properly

TABLE VII.—RELATION OF DURATION OF DISEASE TO MORTALITY INCIDENCE

Deaths Days Post-operative	Days—Duration of Disease										
	1	2	3	4	5	6	7	8-10	11-14	Over	Total
1	2	1	2	2	1		2	1	2	2	15
2 to 5		1	3	2	2	2	3		2		15
6 to 10	2			2	2		1	1		2	10
Over 10	1	1	1	2	1		2			7	15
											2*
Total	5	3	6	8	6	2	8	2	4	11	57

*Not operated upon.

there is no danger of waterlogging the patient. The perineal tube is left in place for from 5 to 7 days. Occasionally after its removal, chills, fever, and signs of urinary sepsis develop, and if these do not immediately subside, indicate the necessity for replacement of the drainage. Many patients have been given hot tub baths in a dilute permanganate solution (1:10,000) as soon as they can be safely transported. This not only makes for cleaner wounds but stimulates the repair process. If strictures have been cut, the passage of sounds is begun from 7 to 10 days after operation and continued every 5 to 7 days. Scrotal regeneration is rapid and complete. (Figs. 9 and 10). Penile cutaneous repair has been accelerated in some cases by the use of Thiersch grafts (Fig. 11).

Postoperative complications are predominantly those of sepsis and renal insufficiency. Seven patients died of pneumonia, 4 of cardiac failure, and 1 of paralytic ileus. Fifty-eight of these 135 patients died (2 before operation could be performed), a mortality of 42.9 per cent. A fourth of these deaths (15) occurred within 24 hours postoperatively (Table VII). All but 3 of this group had had the phlegmon for 3 days or longer. A study of Table VII indicates that of 17 patients who had had this lesion for less than 3 days, 8 eventually died and of 71 suffering from 3 to 14 days, 36 died—a mortality of substantially 50 per cent in each group. Of 12 patients known to have had phlegmon over 2 weeks, 11 died. Therefore, the earlier the patient can be operated upon, the more favorable will the ever grave prognosis become.

SUMMARY

Because periurethral phlegmon is a rapidly fulminating infection which kills nearly half of its victims, the early recognition of the nature of the lesion together with the immediate institution of proper treatment is of prime importance. The prognosis rests not only upon the virulence of the invading organisms, the site and duration of the disease, but upon the degree of renal damage which has occurred. Treatment is surgical, radical, and is always an emergency procedure. Delay and meager incisions are fatal. Free drainage must be afforded the urinary bladder and the involved cutaneous tissues. Genital cutaneous repair is rapid and satisfactory. When stricture has been demonstrated, periodic dilatation with sounds must be employed faithfully after operation, according to the usual custom for all urethral strictures. Such treatment constitutes the only prophylaxis against future phlegmons.

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THE INCIDENCE OF STRICTURE OF THE URETER[1]

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RECENT articles concerning the incidence of stricture of the ureter found at necropsy would lead one to believe that they are common, the percentage of incidence varying from 3 to 90. The material studied, however, has been too limited both as to number and selection of cases to afford a comprehensive survey of the question. A close analysis of the cases reported shows that many of them are not confined to the type of stricture which urologists have recently described clinically. In order to determine the incidence of strictures that cause obstruction to the introduction of a catheter or sound, as well as of the so-called wide stricture, further investigation of a large series of ureters removed at necropsy seemed necessary. To attain even approximate accuracy in such analysis it seemed to us that material available in a general hospital, where all types of diseases occur at all ages, should be studied. We therefore instituted a series of postmortem examinations and are reporting our observations so far as they have been carried out.

Fig. 1. Ureterocele in the presence of a congenitally small meatus, hydro-ureter and nephrosis in upper half of duplicated kidney.

One of us (Frater) completely removed both kidneys and ureters in 93 unselected cases at necropsy. The organs were inspected *in situ* and then removed either intact or with the kidneys detached above the ureteropelvic juncture. Forty-eight of the subjects were males and 35 were females. Each decade of life up to the eighth was represented, including 7 in the first decade.

Roentgenograms were taken of the kidney and ureter in 14 cases and of the ureter alone in 32. The ureter was filled by gravity with a column of fluid (30 per cent sodium bromide) held 37.5 centimeters above the level of the ureter.

After the length of the ureters had been noted, olives attached to a flexible steel guide were passed up the ureter. The size of the olive causing obstruction, and the site of the obstruction in terms of distance in centimeters from the ureterovesical orifice, were ascertained. In the passing of the bulbs care was taken not to use too much pressure. The bulbs were passed from No. 8 French up to the size producing obstruction in sequence. Thus, if obstruction to a No. 14 French was noted, it indicated that a No. 13 French bulb did not meet with obstruction. A section of the ureter about 1 centimeter in length was removed from the site of obstruction and six sections were made through this area after it had been embedded in paraffin. The sections

[1] Read before American Association of Genito-Urinary Surgeons, Washington, May, 1928.

Fig. 2. Roentgenogram showing stricture of the middle portion of the left ureter, with dilatation of the ureter and pelvis.

Fig. 3. Dilatation of upper half of right ureter, with pyelectasis. Stricture was not present on microscopic examination.

were stained with hematoxylin and eosin and van Gieson's stain for fibrous tissue and were then examined microscopically.

It should be stated that of the 93 subjects studied 40 were embalmed and 53 were not. Of those that were embalmed, none was examined more than 10 or 12 hours after death, most of them being examined 4 or 5 hours afterward; the postmortem changes in the tissues of the ureter would, therefore, be comparatively insignificant. Furthermore, it was noted that there was not much difference in the data obtained from ureters that had been embalmed a few hours than from those that were not embalmed. Although the absence of muscle tone or reflex spasm present in the living subject might make a difference in the size of the sounds passed, still it would hardly affect the data obtained as to the comparative diameter of the ureter in various areas.

The average size of the bulb that met with obstruction was between No. 12 and No. 13 French. There was little difference in the sexes or in the embalmed and unembalmed tissue. In four male subjects bulbs varying from No. 16 to No. 20 French were not obstructed in one ureter. In one male subject obstruction was not met in either ureter to No. 19 French. In three female subjects obstruction was not met to bulbs varying from No. 18 to No. 19 French in one ureter, and in one female subject a No. 20 French was not obstructed on either side. It is evident that the caliber of the normal ureter is far from uniform. It is not unusual to observe a normal ureter which will not permit a bulb larger than No. 9 to be introduced without obstruction. On the other hand, an equally normal ureter may permit a No. 19 or No. 20 bulb to pass easily throughout. The fallacy of endeavoring to determine areas of abnormal constriction by means of measuring the caliber by a bulb, when the normal limits are so variable, is obvious. The size of the bulb which may be introduced without obstruction in the normal ureter varies from No. 8 to No. 20. Forty-four per cent of the obstructions were within the first 2 centimeters of the ureter. This included the intramural portion and that immediately adjacent in some cases. The next most frequent site of obstruction was from 2 to 4 centimeters from the bladder and composed 17.5 per cent. Most of the obstructions in these two groups correspond to the site of the third physiological zone of narrowing. In the first 6 centimeters, therefore, 68 per

Fig. 4. Great increase in thickness of ureteral wall with increased fibrous tissue and large lumen, which admitted a No. 20 French olive with the greatest ease (× 18).

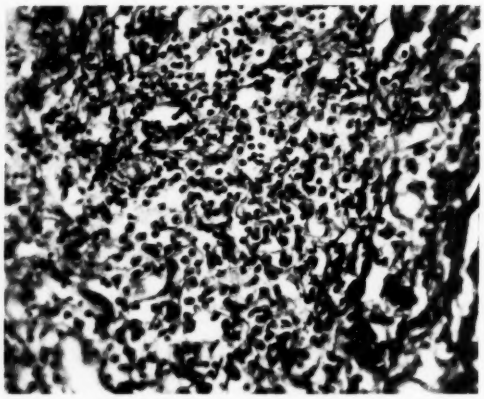

Fig. 5. Photomicrograph of section taken from the same case as that shown in Figure 4. Lymphocytic collections (× 200).

cent of the obstructions occurred. According to some observers, this corresponds to the site at which stricture is most frequently diagnosed. The area, 6 to 12 centimeters, covers the second physiological zone of narrowing, and obstruction was met there in 17.5 per cent of the ureters.

CRITERION OF STRICTURE

A stricture is a narrowing, beyond the normal anatomical and physiological limits, of a hollow muscular tube. The question may well be raised: What are the normal anatomical and physiological limits?

If all ureters were of the same caliber and there was an established normal caliber, it would be a simple matter to diagnose a stricture by means of bulbs. Just as the urethra in one individual may be smaller than that in another, so normal ureters in different individuals may vary in caliber. In the same individual one ureter may have a greater diameter than the other.

Gross strictures are readily diagnosed at necropsy. They are characterized by marked localized narrowing of the ureter with dilatation above the site of the stricture. Definite obstruction to small bulbs can be demonstrated in this type of stricture. The roentgenograms will show a localized area of narrowing with dilatation above. Such areas may be congenital or acquired.

Wide stricture. In the absence of definite pathological criteria of the so-called wide stricture it is difficult to give a detailed description of the pathological changes involved. Microscopically a stricture may show: (1) changes in the epithelium, such as destructions; (2) narrowing of the lumen up to complete obliteration; (3) evidence of inflammatory reaction, such as lymphocytic and leucocytic collections in the wall of the ureter; (4) increase in fibrous tissue at the expense of the muscle bundles, or an increase of the fibrous tissue normally present; and (5) hyalinization of muscle.

In congenital strictures absence of inflammatory reaction will be noted; this may also be noted in the acquired strictures if the process is of long standing. The epithelium may be unchanged in a stricture of moderate degree.

In intrinsic acquired stricture of the ureter the constriction is the result of healing of an injury (used in its widest sense), which has taken place not by resolution but by fibrosis. The fibrosis may be either primary or secondary, the primary taking place without antecedent ulceration, the secondary being the method of healing following ulceration. If sufficient fibrosis occurs following the initial injury and contraction of the fibrous tissue follows, a stricture may result. Localized areas of fibrosis may occur in the wall of the

ureter, without as a rule causing diminution of the lumen. These areas of localized fibrosis, although possibly indicative of previous infection, are not diagnostic of stricture.

In determining any abnormal change in the mucosa, fibrous tissue, or musculature of the ureter, one should first become familiar with the variations that are normally present. In the study of a series of normal ureters microscopically in sections stained by fibrous tissue stains, the large amount of fibrous tissue normally present is apparent. It will vary considerably at the same level in different subjects and in the same ureter at different levels, since it increases in amount over sections nearer the ureteropelvic juncture. The width of the mucosa also shows considerable variation at different levels, being much more marked in the upper half of the ureter. Likewise the musculature is variable in different subjects at the same level, and in the lower half of the ureter an outer longitudinal muscle layer is visible, which is absent in the upper half. In different subjects the musculature may be better developed in one ureter than in the other. In the estimating of any abnormality in the mucosa or increase in fibrous tissue, or hypertrophy of muscle, therefore, comparison with normal variations is essential.

In the determining of the degree of obstruction that may be present in a ureter before it should be regarded as due to stricture, several anatomical factors must be considered. As noted, there is a large variation in the size of bulbs that the normal ureter will admit. Recognition of the normal areas of anatomical narrowing is essential. In embryos up to 125 millimeters total length, the ureter is an almost straight tube of even caliber throughout, but slightly later in embryological life three areas of narrowing occur: one at the ureteropelvic juncture, the second where the ureter crosses the linea innominata between the true and false pelvis, and the third just above the entrance to the bladder. Among these areas of narrowing, fusiform dilatations may occur: (1) in the lumbar spindle between the first and second narrowings—the lumbar spindle is present in embryos of a little more than 125 millimeters total length and is abdominal; and (2) in the pelvic spindle between the second and third areas of narrowing —the pelvic spindle develops in embryos of 320 millimeters total length, is inconstant, and may be completely lacking after birth. The lumbar enlargement is never absent in embryos of more than 125 millimeters and in children may taper off gradually above and below, in which case the upper and middle narrow parts are only indistinctly seen and the middle may be absent. In most of the cases in the series, these physiological areas of narrowing and the intervening spindles were noted, although in some the pelvic spindle and in others the lumbar spindle was absent. In a few neither spindle was present.

One cannot conceive of a narrowing sufficient to be labelled stricture without a recognizable increase in fibrous tissue. The converse, however, does not hold. One of us (Braasch) has frequently emphasized the clinical importance of recognizing the existence of "inflammatory or atonic" dilatation of the ureter. Israel, Alksne, Andler, and other observers have referred to such dilatation as a result of atony. With dilatation of this type all the changes in the wall of the ureter which occur as the result of stricture may be present except the narrowing of the lumen. Sections of the ureter will then show an increase in fibrous tissue, degeneration of muscle, lymphocytic collections, degeneration of epithelium, and yet may admit with the greatest ease, bulbs as large as No. 20 French throughout the length of the ureter.

Small collections of lymphocytes or leucocytes scattered in the wall of the ureter were noted; these were not accompanied by any fibrous or degenerative changes in the surrounding tissues or with constriction of the ureteral lumen. The areas were observed in the different layers of ureteral wall, the majority being situated in the submucosa or musculature. The few areas that were noted in the mucosa did not cause other destructive changes or offer resistance to the passage of a bulb. In the absence of the two essential features of stricture, it is difficult to interpret their presence. Although a collection of lymphocytes often indicates an inflammatory process, it cannot always be so interpreted.

Lymphocytes are frequently observed in various organs in elderly adults and may then be a manifestation of the generalized degenerative process of age. Their presence alone never justifies the diagnosis of stricture, even if slight localized fibrotic changes are also present, unless there is evidence of destructive changes in the mucosa and narrowing of the lumen.

In this series of 93 necropsies actual stricture of the ureter was found in only two. Neither of these was of an infectious nature: one was the result of extra-ureteral carcinoma, and the other was congenital. A so-called wide stricture of inflammatory origin was not found in any of the subjects examined. In the first case there was gross evidence of marked compression of the middle portion of the ureter by metastatic carcinoma, with resulting hydro-ureter and hydronephrosis above, and a small stone. Microscopic study of the site of the stricture showed: (1) increase in fibrous tissue, (2) great narrowing of the lumen, (3) atrophy and replacement of muscle bundles by fibrous tissue, (4) collections of lymphocytes in the wall of the ureter, and (5) destruction of the epithelium.

In the second case there was an extremely small ureteral orifice, causing obstruction with resultant ureterocele, hydro-ureter, and hydronephrosis in the upper segment of a duplicated kidney. The meatus was extremely small and a pointed No. 4 catheter was introduced with difficulty. Serial sections made through the opening did not show an inflammatory process. It seems logical to assume that the partial occlusion of the orifice was the result of congenital stricture.

Besides these two cases, there were fourteen which showed evidence of renal or ureteral lesions. Among these there were several showing dilatation of the ureter without etiological narrowing of the lumen. One case showed marked ureteritis without stricture; another, carcinomatous infiltration of the wall of the ureter causing dilatation of the pelvis. In two male subjects definite pyelectasis without ureteral obstruction or dilatation was noted, and in one subject moderate dilatation of both ureters and pelvis, the latter containing sand. In one female subject (primipara) there was marked dilatation of the upper half of the ureter without any evidence of previous or present inflammation and without stricture. In another female subject (multipara) there was bilateral ureteropelvic dilatation without stricture. There were four cases of pyelitis and one of suppurative nephritis without any evidence of stricture in the ureter. There were two cases of ureteral stone, in which the ureter was normal and did not show evidence of stricture on microscopic study. Thirteen cases showed lymphocytic collections in the wall of the ureter without any increase in fibrous tissue or narrowing of the lumen.

ILLUSTRATIVE CASES

CASE 1. A congenitally small meatus. The patient, aged 55 years (tripara), had suffered from stomach trouble for a year. A history of symptoms referable to the urogenital organs was negative except for nocturia, graded 1. Urine contained pus graded 1 (4 cells to the high power field). At operation a duodenal ulcer was excised and appendectomy performed. Death was due to pulmonary embolus. The right kidney showed complete duplication of ureter and pelvis. The ureter from the upper pelvis was tremendously dilated, as was the pelvis. The meatus was pin-point in size, and a large ureterocele was present (Fig. 1). Serial sections through the opening did not reveal inflammatory reaction.

CASE 2. True organic stricture caused by pressure of metastasis. The patient, a woman aged 64 years (quintipara), complained of upper lumbar pain, girdle-like in distribution and continuous in character. A history of urogenital symptoms was negative. Necropsy revealed carcinoma of the head of the pancreas with multiple areas of metastasis. The kidneys, ureters, and bladder were removed intact, and a roentgenogram was made (Fig. 2). The right kidney and ureter were normal, but the left showed an area of marked narrowing 16 centimeters from the bladder. Above this area, the ureter and pelvis were definitely dilated. There was no obstruction to a No. 19 French olive in the right ureter, and the left ureter admitted a No. 5 French catheter but not a No. 8 French bulb. Above the stricture a small calculus was found. Sections through this area showed a greatly compressed lumen and increase in fibrous tissue; carcinoma cells were not found in the wall of the ureter, although they surrounded it. There were several areas of lymphocytes.

CASE 3. Marked changes in the wall of the ureter without narrowing of the lumen. A man, aged 40 years, gave a long history of renal lithiasis. Right nephrolithotomy had been performed 3 years previously. He was uræmic on admission and died shortly afterward. Necropsy revealed bilateral

pyonephrosis with lithiasis. Both ureters were markedly thickened. The right ureter admitted a No. 20 French olive without obstruction. Sections through this ureter at various levels showed a huge lumen and a very thick ureteral wall with increase in fibrous tissue. The muscle bundles showed degeneration and in part replacement by connective tissue. There was marked infiltration by lymphocytes and leucocytes. The epithelium was retained in part and was squamous in character (Figs. 4 and 5). The left ureter showed obstruction to a No. 13 French olive 8.5 centimeters from the bladder. Sections through this area were similar to those on the right side, but with less change.

CASE 4. Ureteral calculi without stricture. A man, aged 52 years, complained chiefly of angina pectoris. A moderate degree of nocturia had been present. Urinalysis showed pus graded 1 (4 cells to a high power field). At necropsy the ureters and kidneys appeared grossly normal. A calculus was palpated in the left ureter 13 centimeters from the ureteropelvic juncture. The right ureter offered obstruction to a No. 13 French olive 11 centimeters from the bladder and the left 8 centimeters from the bladder. Serial sections from this area in the left ureter did not show abnormality, and those from the right showed a few small lymphocytic collections in the fibrous sheath, but were otherwise normal.

CASE 5. Obstruction due to interference with peristalsis without decrease in the size of the lumen. A man, aged 69 years, complained of abdominal pain of 6 months' duration. A moderate degree of nocturia had been present for the last 4 years. Urinalysis was practically negative. At necropsy carcinoma of the right suprarenal gland with metastasis to the mesenteric lymph nodes was found. The left renal pelvis showed dilatation graded 2; the right was grossly normal. The left ureter presented obstruction to a No. 17 French olive 10 centimeters from the bladder. Sections at this point and at various other points in the ureter showed marked infiltration of the ureteral wall with carcinoma cells, without any increase in the fibrous tissue.

The ureter admitted a No. 16 French olive, yet there was dilatation of the pelvis. This case illustrates atony of the ureter from a rare cause: interference with peristalsis from carcinomatous infiltration of the ureter in practically its whole length. This was discovered only on microscopic examination.

CASE 6. Dilatation of the ureter without obstruction. A woman, aged 31 years (primipara), complained of symptoms suggestive of brain tumor. There was a history of occasional nocturia; the urine was normal. Necropsy disclosed a malignant neurofibroma. A roentgenogram (Fig. 3) showed dilatation of the upper half of the right ureter. Obstruction to a No. 13 French olive was encountered on the right 1.5 centimeters from the bladder; a No. 17 French olive was not obstructed above this point. On the left there was obstruction 8.5 centimeters from the bladder to a No. 14 French olive. Sections from the sites of obstruction, as well as at the point of dilatation, were normal.

SUMMARY AND CONCLUSIONS

1. The incidence of lesions in the ureter is greater than has been previously recognized.

2. The infectious origin of stricture of the ureter is not so common as recent articles would infer, as is shown by the fact that stricture of this type was not found in 93 necropsies.

3. The caliber of the normal ureter as ascertained by the passage of bulbs varies from No. 8 to No. 20 French.

4. The most frequent site of greatest anatomical narrowing in the normal ureter is in the first 4 centimeters from the ureteral orifice, which corresponds to the area in which most strictures have been reported.

5. Lack of symmetry in the two ureters was common. In several instances the caliber of one ureter was 50 per cent greater than that of the other, both being normal on gross and microscopic examination.

6. As the caliber of the lumen of the normal ureter varies, it is difficult to recognize a stricture by means of bulbs or sounds larger than No. 9 French.

7. The demonstration of areas of ureteral dilatation, even when they occur proximal to a portion of the ureter with a comparatively small lumen, does not necessarily indicate stricture. The dilatation in such cases may be atonic and the result of intrinsic cicatricial changes in the wall of the ureter.

8. Microscopic areas of lymphocytic infiltration regarded by some observers as indicative of stricture cannot logically be so classified, since they lack all of the gross and microscopic criteria of stricture.

9. That stricture is not necessary to the formation of renal or ureteral stone is shown by two cases of the series in which evidence of a lesion in the ureter could not be found on gross or serial microscopic section.

10. In at least 8 cases in the series there were some symptoms of urinary disturbance and several in which some tenderness was discovered on palpation of the ureteral area. In none of these could any evidence of ureteral stricture be found.

11. In none of the cases was there evidence of the so-called wide stricture. If, as has been claimed, such strictures are common, one must infer that they may exist without leaving any trace of their existence, even in microscopic sections of the wall of the ureter.

12. That stricture of the ureter of infectious origin does occur is recognized by everyone. Although the material in this study is inadequate to determine the frequency of its occurrence, it nevertheless shows that: (1) the incidence of inflammatory stricture is not as great as recent postmortem studies indicate, and (2) the diagnosis of stricture by clinical methods now employed may be inaccurate.

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BILIARY STASIS AS A FACTOR IN THE PRODUCTION OF GALL STONES[1]

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IT has long been assumed, but never proved beyond question, that stasis of bile in the gall bladder leads to the formation of gall stones. However, it has been proved, as everyone knows, that the human gall bladder normally empties almost completely after a heavy meal, and then quickly refills. Without perversion of this function how can gall stones be produced?

An attempt to study the effects of stasis has been made by the method of cutting the common bile duct sphincter, thus preventing refilling of the gall bladder with fresh bile (1). Masses of what appeared to be inspissated bile were thus produced in the vesicle. They varied in consistency; some were jelly-like, others were putty-like, one resembled a solid cast of the gall bladder. A similar hard cast-like structure has since been produced by a somewhat different method (2). Both of these "stones" were formed in gall bladders in which iodized oil had been placed to allow X-ray observation of their motility.

After repeating the experiment of cutting the common-duct sphincter, Copher and Illingworth (3) have concluded that since they obtained no stones when no iodized oil was placed in the gall bladder, the presence of the oil, and not the induced stasis, is the significant factor in the formation of these "stones."

However, the first report of the formation of such masses with the iodized oil in the gall bladder also mentioned and illustrated their formation without it. "In another cat the sphincter was cut, *the gall bladder being undisturbed*.[2] The animal was fed egg yolk and then allowed to fast for several days. This time the viscus was found partly collapsed and filled with a deep green, jelly-like mass which retained its shape when removed. In a third cat likewise treated, the gall bladder contained several smaller soft, dark masses suspended in mucus (Fig. 9); in a fourth, there was a soft stone blocking the cystic duct (Fig. 10); in others, the gall bladder contained only mucus, presumably having emptied itself of bile"(1).

Cutting the sphincter of the common bile duct without the injection of iodized oil in 12 additional cases was followed by negative results, the gall bladder being found after several days or weeks to contain only mucus. In these cases there was obviously no stasis

[2] Italics are used in the quotation for emphasis.

[1] From the Laboratory for Experimental Surgery, University of Rochester School of Medicine and Dentistry, Rochester, New York.

of the bile in the gall bladder. The absence of the sphincter prevents refilling of the gall bladder, but it does not prevent emptying, and evidently the bile remaining in the gall bladder after the operation had been either ejected or displaced by the secretion of mucus. Thus negative experiments may not be of great significance. It is a mistake to assume that because one does not obtain certain results by certain methods such results are unobtainable.

By other methods, hard, black, cast-like structures have been produced in 2 cases. While being filled with iodized oil the gall bladder in a cat was accidentally stripped away from the liver nearly down to the cystic duct. It was replaced in its fossa and the abdomen closed. The next day the vesicle had expelled most of the oil excepting flecks adherent to the walls, which made the outline visible to the X-ray, and had presumably refilled with bile. This shadow form remained constant in spite of feeding for 11 days, except that it decreased slowly in size. This perhaps indicates inspissation. At necropsy the gall bladder was found filled with a very hard, black cast which bore every appearance of being composed of dehydrated bile. This supposition was strengthened by the finding of a normal mucosa in the vesicle (2). The experiment was repeated several times omitting the injection of the oil, but without the expected result.

In another cat the gall bladder was filled with iodized oil and a physiological experiment performed. At the end of this experiment a shadow form similar to that already described was noted, signifying a gall bladder containing bile with a little oil about its sides. Steps were then taken to maintain stasis in the vesicle. When the animal was killed 2 days later the expected result was obtained. The neck of the gall bladder and upper cystic duct were filled with a firm cast-like structure while the body and fundus (about one-half normal size) contained putty-like material.

In these cases the iodized oil may have been a factor in the induction of stasis, it may have altered the mucosa in such a manner as to promote inspissation, or it may have had other effects. This question is being investigated.

Copher and Illingworth (3) performed a series of experiments in which iodized oil was merely injected into the normal gall bladder, and they sometimes found these masses afterward, although no stasis was present. They stated that these experiments seemed to indicate clearly that the iodized oil rather than stasis was the important factor in the production of the so-called "gall stones."

Taking in order from my files 46 records of experiments in which iodized oil was injected into the gall bladder of the cat, I obtain some interesting and indecisive figures. In 34 experiments, running from 1 to 43 days, no masses were found in the gall bladder; in 11 experiments, running from 1 to 8 days, masses were found. The oil may have produced these masses, but it was not a very effective producer. However, in 46 *normal* cats in which the gall bladder was examined after feeding experiments masses were never found.

Copher and Illingworth have also suggested that since cholesterol has not been found a constituent of these masses they bear little relation to human gall stones, but instead result directly from the presence of iodized oil (3). However, it must be remembered that some gall stones contain little if any cholesterol. Furthermore, how shall we explain the formation of masses in the gall bladder where no iodized oil has been injected? (1) It is possible that the iodized oil or the operation of introducing it brought about conditions favorable to the formation of these masses, but in view of the above facts, the conclusion that they "result directly from the presence of iodized oil" (3) seems unjustified.

Undoubtedly the relation of cholesterol to the formation of gall stones is of prime significance. It has never been claimed that stasis is the only factor in gall-stone formation, but it is difficult to see how cholesterol could stay long in a gall bladder which contracts normally after meals. The question of the effect of cholesterol in the gall bladder plus stasis has been investigated. In 14 cats cholesterol mixed with bile was placed in the gall bladder and stasis induced by fasting. After 6 days, single brownish-black masses were formed in the gall bladder in 2 cases. They were 5 to 7 millimeters in the short diameter and rather

oblong; one was firm and the other putty-like in consistency. In another case there was a small mass of white, glistening, semi-solid material strongly adherent to the mucosa of a gall bladder containing bile-stained mucus. In other cases only cholesterin crystals and normal bile were found.

None of these "stones" has yet been examined chemically. There is a possibility, as suggested by Dr. Whipple, that their basis may be blood clot resulting from the operation. Masses have been found which do look somewhat like blood clot, but some of the softer masses when spread out on filter paper produced only a greenish stain. Of course, blood clot could hardly be the basis of the masses formed in "undisturbed" gall bladders.

In the effort, however, to eliminate as far as possible all factors save stasis and concentration of bile in the gall bladder, a new series of experiments was performed. We know that there is at least some stasis in the gall bladder during fasting, and, of course, dehydration should increase the concentration of body fluids. Consequently 9 cats were kept under the influence of barbital, sleeping peacefully, neither eating nor drinking. Two were negative, but in 7 cats which were kept for 5 days to 15 days there were numerous fine particles in the concentrated bile of the gall bladder, varying in size from dust to 1 to 2 millimeters and varying in consistency from semi-solid to solid. No such particles were found in the gall bladders of the 46 normal cats already mentioned, and I do not remember ever having seen them thus, though similar particles are often found in the concentrated "muddy" bile in the gall bladder of patients. No large black masses or casts of the vesicle were produced, however, in any of these experiments.[1]

[1]A number of the hard, black masses and castlike structures formed after the injection of iodized oil or cholesterol into the gall bladder have recently been given the benzidin test with such markedly positive reactions as to leave little doubt that they are composed chiefly of blood clot. It is probable that damage to the delicate vascular mucosa at the time of injection, resulting in hæmorrhage into the lumen, was responsible for these masses erroneously supposed to be stones. But the smaller masses formed under the barbital-fasting method of inducing stasis in the gall bladder gave negative tests for blood.

It has been suggested that intramural infection of the gall bladder may be an etiological factor in gall-stone formation (1, 2, 3). Gall stones have recently been produced by A. L. Wilkie (4) following the intravenous injection of streptococci. Marked thickening of the wall of the gall bladder was induced, but the mucosa was often intact. What more probable explanation than that infection of the wall of the gall bladder induced stasis by inhibition of the musculature, while concentration continued through the activity of the mucosa, resulting in stone formation? (1, 2). Attempts are now being made to induce stone formation through damage to the wall of the gall bladder without infection.

Thus it will be seen that the problem is extremely complex and beset with difficulties and uncertainties. However, it seems to me that on the whole recent studies confirm the old opinion that stasis is an essential factor in gall stone formation. For instance, it has been shown that the human gall bladder normally empties almost completely after meals, and also I have found that after feeding, the gall bladder of the cat will expel quantities of matter in small particles. It would seem, then, that stasis, which means failure of this expulsive function, must be present in order that any material may remain long enough in the gall bladder to be formed into stones. Furthermore, as noted above, masses can be produced in the gall bladder simply by the physiological induction of stasis.

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CLINICAL SURGERY

FROM THE WOMAN'S HOSPITAL, NEW YORK

THE OPERATIVE TECHNIQUE FOR THE REPAIR OF RECTOCELE AND INJURY TO THE PELVIC FLOOR

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A PROPER understanding of the mechanism of the closure of the vaginal orifice is essential in order to appreciate the conditions of impaired function, the result of an obstetrical injury, which we designate as rectocele and laceration of the pelvic floor. A usual idea is that the closure of the orifice is sphincteric similar to that of the anus, while in fact the actual mechanism of the opening and closing of the vaginal introitus is quite different. In teaching we usually compare the mouth and the vaginal orifice.

In the mouth, we have a transverse slit with a fixed upper jaw and a movable lower jaw, the mouth being closed by the raising of the lower jaw against the upper by the masseter muscles. If these muscles were cut, torn, or stretched, there would result an inability to close the mouth, and the lower jaw would hang down.

In the vaginal orifice, we have a transverse slit, with a fixed anterior and a movable posterior vaginal wall constituting the pelvic floor. The orifice is closed by the raising of the mobile posterior against the immobile anterior segment by the levator muscle, as in the mouth, and not by a sphincteric action, in spite of the so called sphincter vaginæ muscle.

Ramifications of the pelvic fascia give support and strength to the levator in the vaginal sulci, where it is ruptured by the advancing fetal head, when it receives the brunt of the strain during the internal rotation in labor, or is torn, frequently in both sulci, by the forceps blades. A condition then produced is similar to a rupture of the fascia lata of the thigh when the strain in standing would fall upon the quadriceps extensor, with resulting tire and ultimate stretching of that muscle. The torn pelvic fascia allows the strain to fall upon the anterior fibers of the levator torn away from its lateral attachments to the rectum and the perineal body, into which the fibers of each muscle decussate. The consequence is ultimate stretching and relaxation, while the result is that the posterior segment of the pelvic floor is not properly lifted because of the elongated or torn levator fibers. The vaginal mouth, therefore, is constantly gaping, and the unsupported vaginal walls with their attached viscera tend to roll down and out. The strength and support of the posterior vaginal wall and rectum reside in the firm barrier formed by the fusion of the two layers of the levator fascia, the fascia of the urogenital diaphragm, and Colles' fascia at the site of the perineum. Should this fascial support of the posterior vaginal wall and rectum be injured, the open vaginal mouth then favors the protrusion or hernia of the anterior rectal wall at the site of the injury, designated a rectocele. Such an anatomical change alters the normal mechanism of defæcation by diverting the direction of the fæcal current so that the anterior rectal and the posterior vaginal wall receive the brunt of the strain with a consequent protrusion which increases until a distinct rectal pouch forms and renders complete evacuation difficult.

Conditions in a rectocele are similar to cystocele. Both are due to an injury of the fascial supports, and their development is accelerated by the patent vaginal orifice following the pelvic floor injury.

In the past, the operation advocated to cure cystocele and rectocele was the shutting of the vaginal mouth by an operation based on the Emmet principle, and the taking up of the slack or excess of vaginal tissue by a superficial denudation of the mucosa and the approximation of the edges. This apparently produced a good immediate result, but the prolapsed bladder and rectum were simply thrown into folds, which the daily exercise of their functions soon obliterated, and the results were evanescent.

In recent years, the cystocele problem has been well worked out. B. E. Hadra first and then M. Saenger urged a more radical procedure to insure permanency and showed that the bladder must be

completely separated from the vaginal wall, as well as the uterus, and shifted to a higher plane in the pelvis and that the fascial opening must be repaired, and finally the excess of the vaginal wall resected.

In rectocele we have a true hernia or prolapsus of the rectum perfectly analogous to cystocele. The bowel also becomes enlarged and pouched until there is an increase in the size of the gut, similar to that of the bladder in cystocele.

It is obvious that as a rectocele parallels cystocele, we ought to apply the identical principle which has proved so successful in vesical prolapse, and this we have done with uniform success for many years.

The operation consists in the complete separation of rectum and posterior vaginal wall, as far up as the cul-de-sac of Douglas, the sliding of the loosened rectal pouch high up along the vaginal wall, the fastening of it there with a suture, and closure of the fascial opening. By this the denuded rectum is drawn up and secured and made to adhere to the upper undamaged posterior vaginal wall well *above* the site of the former rectocele. This procedure we have designated as a "rectopexy."[1]

A perineorrhaphy follows in the form of a muscle operation. By approximating the anterior levator fibers, the muscle barrier thus formed acts as a dam to prevent the recurrence of the rectocele, in addition to furnishing an effective restoration of the vaginal orifice. The perineorrhaphy described has been done in its essentials by the author since May, 1908, with slight modifications from time to time.

When we recall the normal decussation of the anterior fibers of both levators in the perineum, any objection to this type of operation, as anatomically incorrect, is not valid.

The improved technique as we now do the operation at our clinic at the Woman's Hospital is as follows:

The labia minora are drawn out of the way and sutured to the skin. A gauze sponge on a sponge holder is inserted in the rectum as a guide. The introitus is opened wide with a Friedman retractor which catches each posterior caruncle just below the orifices of Bartholin's glands, care being taken not to occlude them. A third forceps is attached to the posterior vaginal wall in the median line, marking the crest of the rectocele. While traction is made on these tenacula, the resulting triangle is outlined with a scalpel. This area represents the excess vaginal wall to be removed subsequently; the marking of its boundaries at the outset greatly facilitates its accurate removal as a later step (Fig. 1).

With blunt scissors, the base of the triangle is dissected free from side to side, and the superficial and fused fascial structures cut through. By blunt dissection the gauze-covered finger opens up the line of cleavage between the side of the rectum and the levator muscle in each sulcus, the finger penetrating deeply between the muscle and its superior layer of fascia which is also attached to the rectum and the under surface of the vaginal sulcus. This dissection ought freely to expose the anterior fibers of the levator as well as its superior surface (Fig. 2).

The rectum is next separated from the posterior vaginal wall well above the area of the vagina outlined for removal, by the insertion and pushing up the line of cleavage of closed blunt-pointed scissors, which are then opened wide and withdrawn while open. The sponge and forceps in the rectum furnish a guide as to the path of safety. A wide space is thus opened up between the rectum and vagina well above the site of the rectocele (Fig. 3).

The levators are now freely exposed and the rectum separated from the vagina; the layer of pelvic fascia covering the superior surfaces of the levators (rectovesical fascia) and attached to the sides of the rectum and to the under surface of the vaginal sulci upon separation from the muscle forms partitions which divide the dissected area into three spaces. Curved clamps are placed on these fascial partitions close under the vaginal wall, extending upward about 1.5 centimeters, and the fascia severed from its vaginal attachments (Fig. 4).

A No. 2 tanned catgut suture is passed through the vaginal wall in the midline, well above the site of the rectocele, is brought down between vagina and rectum and passed through the lower margins of the fascial stumps grasped by the clamps, and returns to pass back through the vaginal wall near the first point of entrance.

When this suture is tightened and tied it obviously draws the mobile rectum upward well beyond the limit of the subsequent resection of the vagina. Thus the denuded rectum is carried up and placed where it will adhere firmly to the upper undamaged third of the posterior vagina above the former site of the rectocele. We designate this procedure as "rectopexy" (Fig. 5).

The dilated part of the vaginal wall which entered into the formation of the rectocele is then cut away along the lines of the incision outlined at the beginning of the operation (Fig. 6), and the cut edges of the vagina are sutured with

[1] Surg., Gynec. & Obst., 1913, xvii, 361.

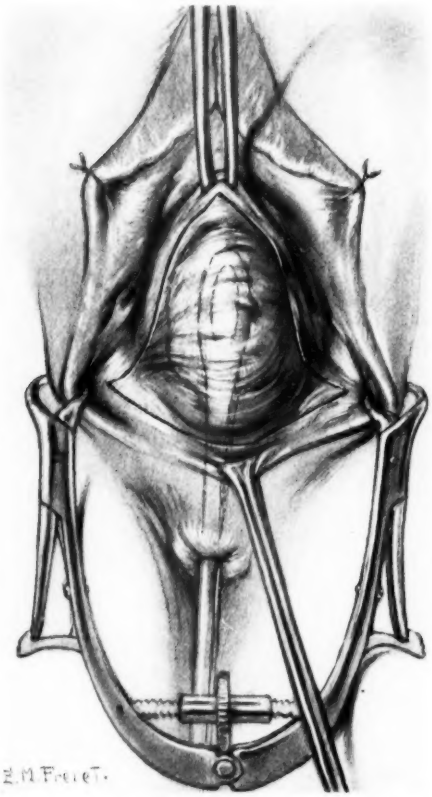


Fig. 1. Sponge in rectum outlines rectocele. Friedman retractor opens introitus widely exposing vaginal sulci. Triangular area of vaginal wall which is to be removed is outlined with a scalpel.

interrupted tanned catgut sutures, taking care to include the two fascial stumps in the upper sutures so as to insure the closure of the space between them (Fig. 7).

The anterior margins of the levators are then grasped with sponge forceps, drawn toward the midline (Fig. 8), and sutured together with interrupted catgut sutures (Fig. 9A). The effect of this approximation of the levators is immediately apparent, the shortening of the muscles lifting up the relaxed pelvic floor and forming a strong barrier to further descent of the rectum.

Care must be taken not to overcorrect the elongation of the levators by placing the approximation sutures too high. Criticism is sometimes made against this type of operation that it produces an objectional band across the introitus

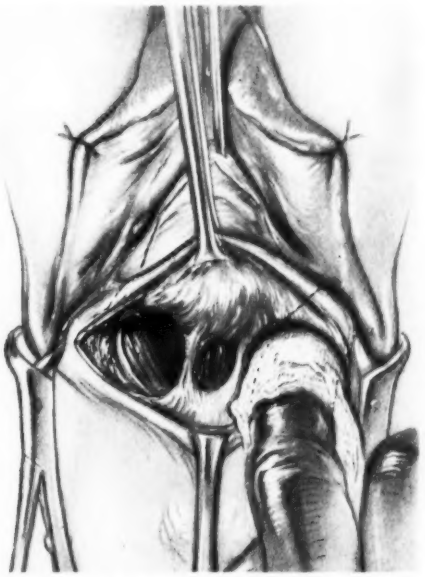


Fig. 2. Superficial and fused fascial structures cut through and levator muscle and rectum separated in each sulcus by blunt dissection with gauze-covered finger. Anterior fibers of levator freely exposed.

Fig. 3. Rectum separated from vaginal wall well above area outlined for removal. Blunt-pointed angular scissors inserted in line of cleavage while closed, then opened widely and withdrawn while open. Sponge forceps in rectum is a guide to the path of safety.

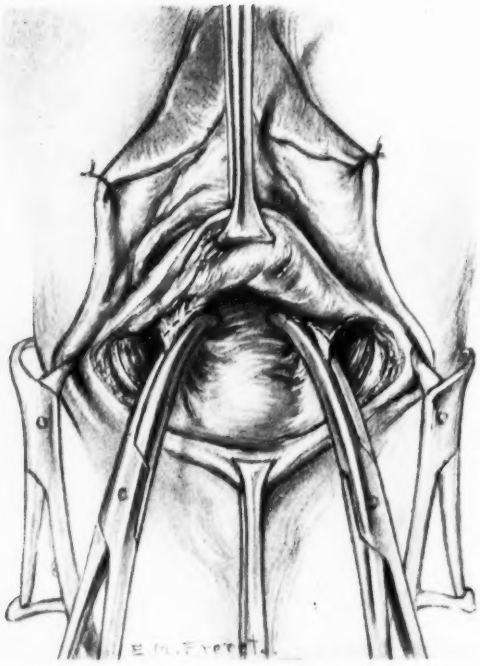


Fig. 4. Fascial structures separating rectum from levators clamped close to vaginal wall and upward for 1.5 centimeters, and cut away at site of dotted lines.

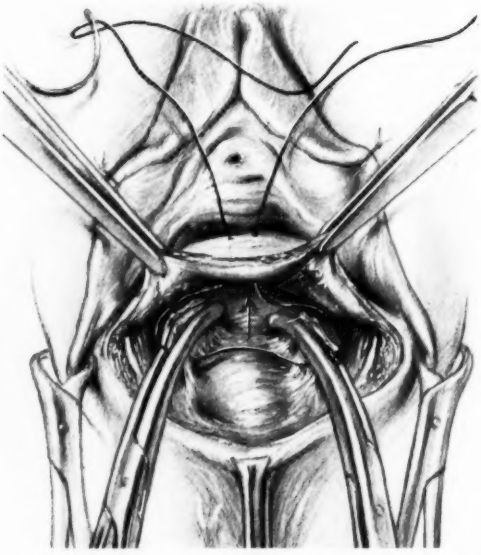


Fig. 5. Rectopexy. A suture is passed through vaginal wall in midline well above position of rectocele and is brought down between vagina and rectum and passed through lower margins of fascial stumps and returned through vaginal wall close to point of origin. Clamps removed and suture tightened and tied. Mobile rectal pouch thus drawn upward and fastened to undamaged vaginal wall above site of rectocele.

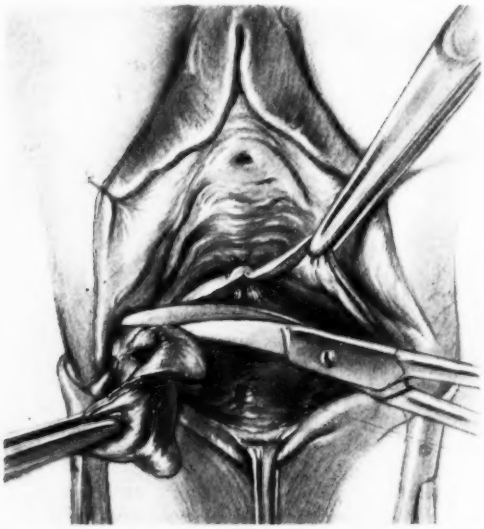


Fig. 6. Rectopexy stitch tied and dilated vaginal wall cut away along incision outlined at beginning of operation.

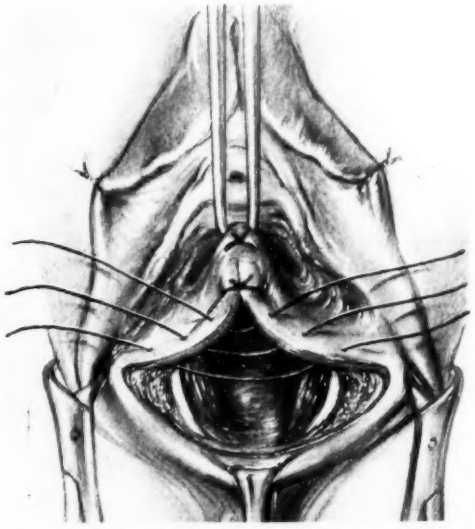


Fig. 7. Cut edges of vagina sutured with care to include the two fascial stumps in the upper sutures.

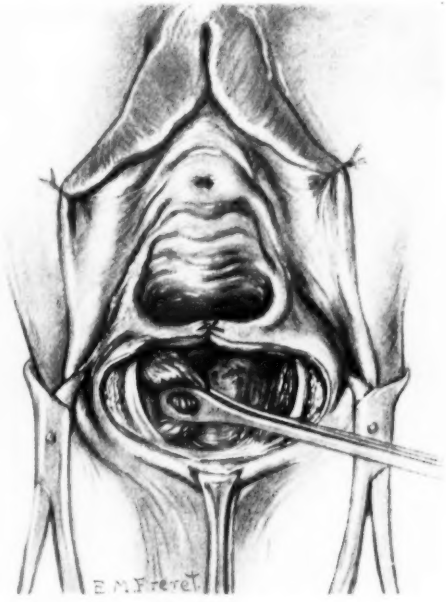
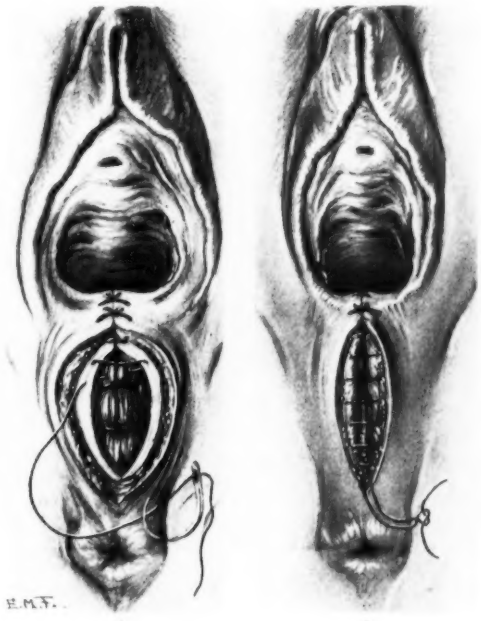


Fig. 8. Anterior margins of levators grasped with sponge forceps and drawn toward midline.



A B

Fig. 9. A, levators sutured together with interrupted catgut sutures. Sharp edge of Colles' fascia seen on each side of wound next sutured with continuous suture, which at its origin is passed wide and deep to include fused fascial structures at this point. Suture also catches united levators. B, skin margin then closed with a subcuticular tanned catgut suture and end tied to fascial stitch. The knot disappears between margins of the incision.

which may be tender and troublesome. If the operation is properly done, there will be no such criticism as it is always an indication of overcorrection. It must not be forgotten that the complete relaxation of the muscles, due to anæsthesia, is not the condition existing when the muscles have regained their full tone.

The sharp edge of Colles' fascia on each side of the wound close to the united levators is sutured with a continuous No. 1 tanned catgut suture. At its origin this suture is passed wide and deep to unite the fused fascial structures of the urogenital diaphragm, levator fascia, and Colles' fascia; it also catches up the united levator muscles and is half hitched at its termination and left long to tie to the subcuticular suture (Fig. 9A).

The skin margin is then closed with a subcuticular suture of No. 1 tanned catgut on a fine needle and the end tied to the fascial stitch, (Fig. 9B); the knot disappears between the edges of the incision.

FROM THE UROLOGICAL CLINIC, PRESBYTERIAN HOSPITAL

DIVERTICULUM OF THE BLADDER

HERMAN L. KRETSCHMER, M.D., F.A.C.S., CHICAGO

DIVERTICULUM of the urinary bladder is a condition which until comparatively recent years has received but little if any clinical recognition, in spite of the fact that the condition has been known and recognized for many years. Morgagni (1683–1771) was one of the early writers and probably the first to recognize its true nature. Honstet, Bonet, Tenon, and Chopart were also early writers on the subject. Up to 1906, only 5 cases had been reported in the literature of the United States. The scarcity of case reports was not due to the fact that the condition was not known, but to the fact that the special instruments necessary for its diagnosis were not available. Today with the perfection of the cystoscope, the use of the roentgen-ray, and the more widespread use of cystograms, diverticulum of the bladder is recognized by careful investigators; hence, the number of patients operated upon has greatly increased, as has likewise the number of cases reported in the literature.

In the diagnosis of the diverticula, four procedures may be used: (1) cystoscopy, (2) cystography, (3) contrast cystograms, and (4) the coiling of the ureteral catheter in the diverticulum.

In every case in which the patient has residual urine, either with or without infection, the possibility of a diverticulum should always be thought of during cystoscopic examination. When the opening of the diverticulum is large, it can readily be recognized with the cystoscope. But in the presence of a severe cystitis a diverticulum may be overlooked. Cystography often reveals the presence of an overlooked diverticulum. Cystograms should be made in two diameters, and there is no objection to making stereoscopic roentgenograms. To determine the size of the diverticulum a ureteral catheter may be coiled up in the sac, the sac injected with bromide solution, and the bladder cavity injected with air.

Small cellules or saccules call for no special treatment. It is always best to remember that the presence of a diverticulum means obstruction and that when the obstruction is removed these small diverticula cause no further trouble. The larger diverticula, however, call for surgical removal by complete excision; nothing short of this must be done if permanent relief is to be attained.

Two methods of removal are in use: the extravesical and intravesical.

Since a diverticulum is always associated with obstruction, and since a certain degree of infection is frequently present, preliminary treatment may be indicated. At times an indwelling urethral catheter may be used and daily bladder irrigations instituted. Silver nitrate or potassium permanganate are drugs commonly used for this purpose. I have repeatedly seen an appreciable diminution in the size of a diverticulum under catheter drainage of the bladder.

A two-stage operation may be done. At the first operation suprapubic drainage is instituted, and at the second operation the diverticulum and the obstruction are removed. Some surgeons advise stretching the neck of the diverticulum to aid in the drainage of the sac and recommend that this be done at the time that suprapubic drainage is instituted. The importance of clearing up the infection cannot be overemphasized.

In view of the fact that diverticula are often associated with residual urine and infection, a very careful study of the renal function should be made, and operation should not be attempted until function has become stabilized.

The operation may be begun with local anæsthesia, which is followed by some form of inhalation anæsthesia.

A median suprapubic incision is made through which the bladder is exposed and as much of the peritoneum as necessary dissected from the bladder, depending upon the location of the diverticulum. The bladder is opened with a wide incision to insure a good view of the inside. The bladder is carefully inspected to locate any diverticula which may have been overlooked with the cystoscope. At this time one may decide upon either the extravesical or intravesical method of resection.

EXTRAVESICAL RESECTION

The opening of the diverticulum is brought into view and the cavity packed tightly with a gauze strip 2 inches wide. This converts the sac into a semisolid tumor and makes the dissection

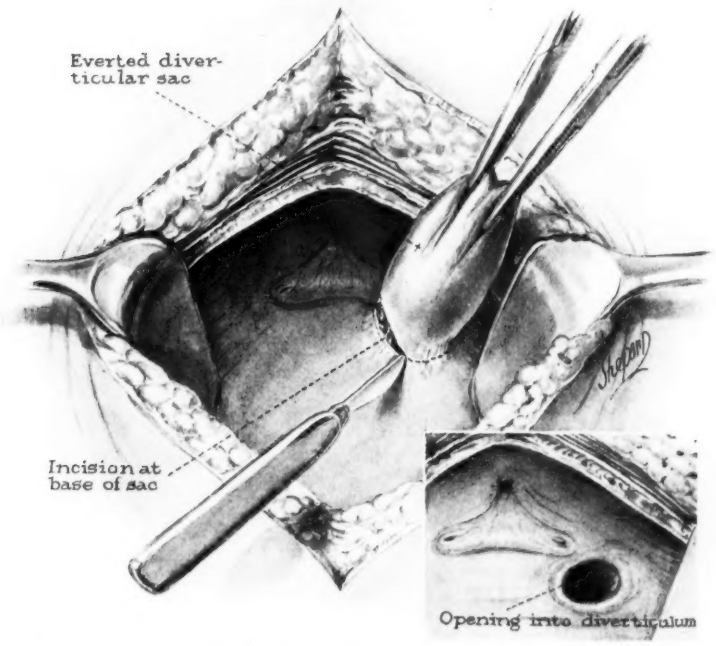


Fig. 1. The sac has been everted by means of artery forceps and an incision made at the base of the sac. The insert shows the opening of the diverticulum near the right ureteral orifice.

of the sac outside the bladder down to the neck a relatively simple procedure. The sac is severed at its attachment to the bladder and the resulting opening closed with catgut sutures. It is most important that this closure be made with good firm apposition of the muscular wall. A drain is carried down to the suture line and the bladder closed in the usual way. As far as I know, Lower was the first to pack the diverticulum with gauze as an aid in its removal.

INTRAVESICAL DIVERTICULECTOMY

Whether one does an intravesical or an extravesical diverticulectomy is merely a matter of choice. I have always used the intravesical method. After the bladder has been opened and the diverticulum located, the next step is the intravesical eversion of the sac. The sac may be everted by suction—as advocated by Young—or by means of artery clamps. I have always preferred the use of clamps. If small, the opening of the diverticulum may be enlarged by dilatation with forceps. The sac is then grasped with clamps and gradually everted. After the inversion, it is well to determine again its exact relationship to the ureter. A circular incision is made around the neck of the sac, after which the sac is separated by blunt dissection with a gauze sponge or the handle of a scalpel. At this stage, large vessels may be encountered; they should be clamped and ligated.

The opening in the bladder is closed with a row of catgut sutures placed on the inside of the bladder. The resulting cavity outside the bladder is drained with a cigarette drain and the bladder closed in the usual way around a suprapubic drain. When the ureter opens into the sac or is situated at the margin, the incision along the neck of the diverticulum should be made at a safe distance from the ureteral orifice so as to avoid injury to the closing mechanism of the ureter. During the dissection of the sac, great care should be exercised to avoid injury to the ureter itself; and to safeguard against any possible

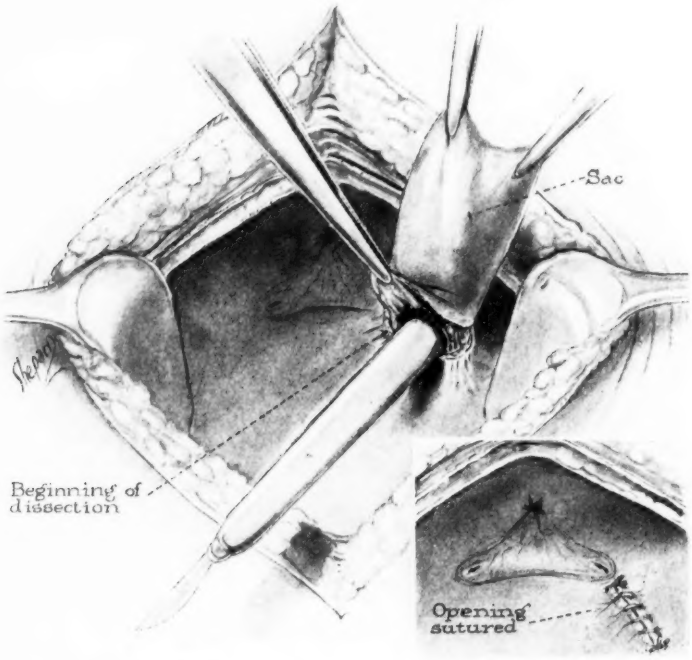


Fig. 2. The dissection is begun by means of the handle of the scalpel. The insert shows the final closure with sutures.

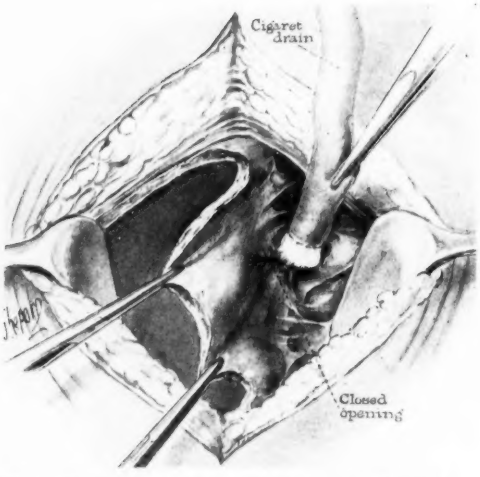


Fig. 3. Insertion of a cigarette drain down to the opening.

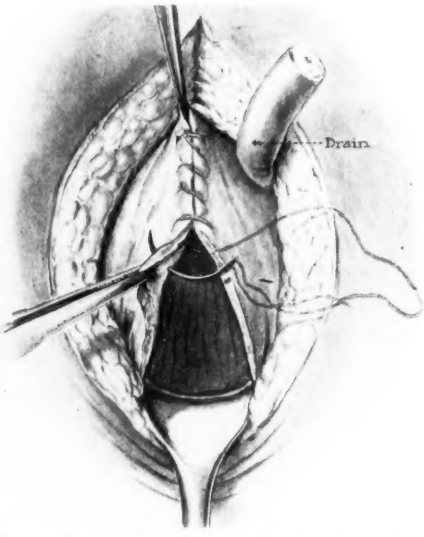


Fig. 4. Closure of the bladder with a running stitch.

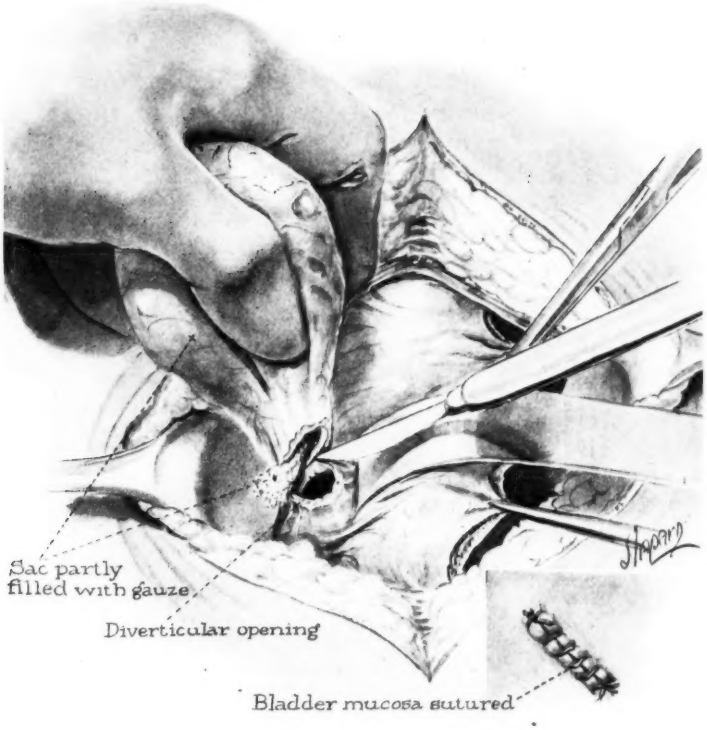


Fig. 5. Extravesical removal of the diverticulum. The sac has been partly filled with gauze to aid in the dissection.

injury during the dissection, a ureteral catheter may first be passed up the ureter.

The treatment of the obstructing lesion, whether by prostatectomy, resection, or by a median bar punch, must not be overlooked, and can be done at the same time or at a later date.

The removal of the suprapubic catheter or drain depends upon the degree of infection still present and the rapidity with which it clears up. As a rule it can be removed on the third day after operation, at which time an indwelling catheter is placed in the urethra.

TUBERCULOSIS OF THE CERVIX UTERI

With a Report of Two Cases, One Probably Primary in the Cervix

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TUBERCULOSIS of the cervix uteri is a rare lesion. It occurs in probably no more than 3 or 4 per cent (Kelly) of cases of pelvic tuberculosis. It has been suggested that this immunity is due to the tissue resistance of the stratified squamous epithelium of the vaginal portion of the cervix and also to the bactericidal quality of the cervical secretion which Menge was unable to infect with various pathogenic organisms. Tubercle bacilli have never been demonstrated in vaginal secretions.

Tuberculous disease of the cervix was originally described by Raymond in 1831, and Virchow reported a case in 1853; since then a scanty literature has accumulated. Moore stated in 1919 that approximately 20 cases of primary and 150 of secondary tuberculous lesions of the cervix had been reported, and that Eden, Lockyer, and Williams had found that one of 600 women with pulmonary tuberculosis had tuberculosis of the cervix.

Involvement of the cervix with tuberculosis is either a bloodstream infection or is an ascending infection from a primary genital lesion. Primary infection of the cervix has been found in women whose husbands had pulmonary tuberculosis, the infection taking place both by genital contamination through tuberculous sputum, infection of hands, etc., and by transmission of the tubercle bacilli from a tuberculous epididymitis. It has been fairly well established that tubercle bacilli may pass through normal capillary membranes, hence genital infection is possible in the female from a male with pulmonary tuberculosis but without demonstrable genital lesion. There is sufficient evidence to make us believe that many cases of genital tuberculosis in women are transmitted by coitus. Anatomically, tuberculous lesions of the cervix have been classified as miliary, interstitial, papillary, and ulcerative. In the majority of early cases the lesion is hypertrophic, proliferative, or vegetative in type, whereas in the more advanced stages true tubercle formation and ulceration with loss of tissue is a pronounced feature.

It seems likely that anatomical classifications are really descriptive of varying stages of the same pathological process. Microscopically, there is tremendous variation in the picture, there being hyperplasia of the glands, granulation tissue, degeneration, and caseation, all occurring in various portions in the same section. Giant cells vary in number as well as do typical tubercles, and the irregularity of the glands, particularly in the early stages, produces some resemblance to carcinoma. There is normally little difficulty about making a diagnosis by microscopic examination, but grossly the picture is often confusing, and the symptoms are extremely variable and indefinite. Malaise and occasionally fever occur. Amenorrhœa has been reported as a symptom in approximately 50 per cent of the cases, and leucorrhœa is a common early symptom. Slight bleeding after coitus is common, but blood-stained purulent discharge has been described as a typical finding in contradistinction to the more watery discharge of carcinoma. In tuberculosis, tissues tend to be softer than in carcinoma, lacking friability, but sometimes the tissues are extremely tough during stages of extreme infiltration. The ulceration and firm fixation of the cervix, however, are very suggestive of cervical neoplasm, and cases are almost certain to be mistaken for carcinoma on pelvic examination even with the most careful inspection, which happened in our first case (Case 1). Recently two cases have come under our observation.

Case 1. Mrs. G. W., aged 46 years, colored, IV-para. The patient came into the hospital complaining of an excessive bloody and watery discharge of 4 weeks' duration. She was seen by her family doctor, who suspected carcinoma of the cervix and referred her to the hospital. Vaginal examination revealed a relaxed outlet. The cervix was large, apparently softened, and patulous. The external os was irregular and ulcerated. The ulcerative process, however, did not involve the vaginal mucosa but seemed rather to extend up into the cervical canal. Trauma incident to curettage did not produce excessive bleeding (Fig. 4). Dilatation was unnecessary. The uterus was large and boggy but movable, and there were bilateral masses palpable. Two sections were removed from the cervix for biopsy. The clinical diagnosis was carcinoma of the cervix.

Histological examination. The squamous epithelium of the cervix shows ulceration and degenerative changes on the surface with proliferation in the lower strata and mitotic figures. In the submucosa are some masses of epithelioid cells. There several giant cells are seen. There are large numbers of wandering cells of various sorts, leucocytes, and areas of hæmorrhage and necrosis. Diagnosis: infectious granuloma of the cervix, probably tuberculous. The physical examination was entirely negative except for the pelvis. Considering the patient's age and the inflam-

Fig. 1. Case 1. No. 117492. Photomicrograph showing ulceration and degeneration near the edge of the cervix where well preserved epithelium is seen. Numerous giant cells are seen in typical tuberculous granulation tissue.

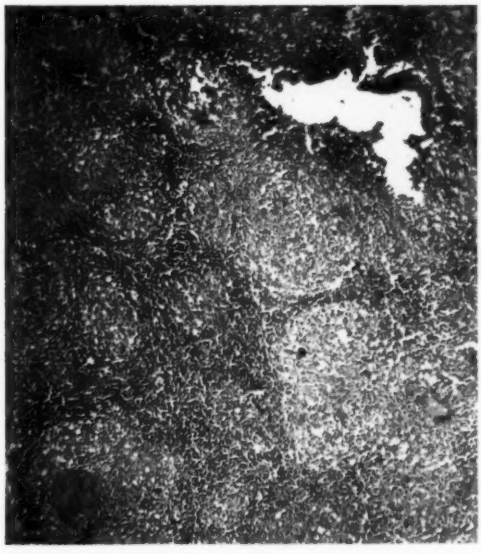

Fig. 2. Case 2. No. 122704. Photomicrograph showing tuberculous granulation tissue with giant cells and well marked tubercle formation, showing also the remains of a cervical gland. Tubercle formation is the feature of the histological picture in this case as compared to the other. Although tubercle bacilli could not be demonstrated in the tissue, the diagnosis of tuberculosis is justified on histological grounds.

matory character of the lesion, surgical removal seemed to be the method of choice.

Operation. When the abdomen was opened, relatively few but very tough adhesions were found binding down both tubes and ovaries. Total hysterectomy was performed, the tubes and ovaries being removed (Fig. 3). The uterus contained a single intramural fibroid the size of a hen's egg. The myometrium was fibrotic. On the surface of the uterus there was evidence of partially organized exudate. The cervical canal was filled with polypoid irregular red masses varying in size up to 1 centimeter in diameter. The consistency of the tissue was firm and rubbery, not hard or friable. The endometrium was smooth and uniform. There were no gross abnormalities. The right ovary was small, necrotic, and covered by fibrous adhesions. The other ovary was similar in appearance and contained a small corpus luteum. The cyst contained a small, circumscribed, white caseous area in the cortex. The tubes were sealed off, thickened, and covered with fibrous adhesions. Their surface was studded with small white tubercular nodules.

Histological examination. In certain areas of the section there was marked hyperplasia of the cervical glands with typical Nabothian cyst formation; elsewhere there was diffuse, round cell infiltration, central necrosis, and typical tubercle formation (Fig. 1). There were numerous giant cells visible. The endometrium presented a picture of diffuse infiltration with disappearance of the uterine glands, a few remnants of which could be seen. There were numerous clumps of round cells forming typical tuberculous granulation tissue. The tubes contained tubercles and giant cells. There was almost complete disappearance of the plicæ surrounding the lumen of the tubes, the epithelium of which was intact in some places.

The patient made an uninterrupted recovery and has remained in good health over a period of 2 years.

Case 2. Mrs. E. C., colored, aged 22 years, nullipara. This patient was admitted to the hospital on January 24, 1928, complaining of amenorrhœa and headache of several weeks' duration. Leucorrhœa was a marked symptom. No bleeding was observed at examination. Vaginal examination revealed a marital outlet. There were several venereal warts on the vulva. The cervix was irregularly ulcer-

Fig. 3. Case 1. No. 117492. Specimen obtained at panhysterectomy. Both tubes are studded with white tubercular nodules.

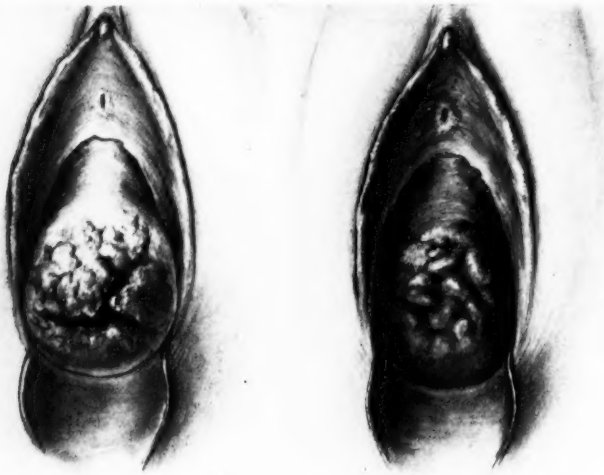

Fig. 4 (left). Case 1. No. 117492. Drawing of the cervix as seen at biopsy when a diagnosis of carcinoma was made.

Fig. 5. Case 2. No. 122704. Gross appearance of the cervix when biopsy was taken. Clinical diagnosis of tuberculosis of the cervix was made in this case. The patient had never menstruated.

ated, tough fibrous areas alternating with sulci. It was markedly hypertrophied, and the consistency did not quite suggest carcinoma (Fig. 5). The uterus was not easily made out and was very small. There were no lateral masses. Carcinoma could not be ruled out in gross examination. Physical and roentgen-ray examinations of the chest were entirely negative. The blood count showed red blood cells, 4,100,000; white blood cells, 8,840; hæmoglobin, 75 per cent. Examination of the urine was negative. Wassermann, 4+. A small section was excised for microscopic examination.

Histological examination. Scattered throughout the stroma of the cervix were typical giant cells of the Langhans type. There was very little reaction around the giant cells although a few showed some lymphocytes surrounding them (Fig. 2). Fibrosis of the stroma was somewhat marked, and there was hyalinization of the bands of connective tissue. The glands were widely dilated and filled with a basophilic amorphous substance. The lining of the glands was the normal columnar type in general with occasionally a transition to a more rounded polygonal type. In other sections were well marked focal areas of necrosis surrounded by slight infiltration of epithelioid cells. The giant cells were more numerous, and throughout the stroma was diffuse infiltration of lymphocytes with a few polymorphonuclear leucocytes. Diagnosis: diffuse tuberculosis of the cervix uteri.

The patient was placed on vigorous antiluetic therapy. The lesion of the cervix has shown some slight but apparent improvement. Subsequent observation has shown no improvement with antiluetic therapy. The patient has not consented to operation. There is no evidence whatever of tuberculosis elsewhere in the body, and we are led to believe the second case to be an example of primary tuberculosis of the cervix uteri. It has been impossible to induce the patient's husband to come to the hospital for examination in the hope that the presence of a tuberculous epididymitis would establish the diagnosis of primary tuberculosis of the cervix as almost certain.

CHARACTER OF LESIONS

When freed from superficial discharge these lesions present a picture which is at once ulcerative and proliferative in character. It suggests the vegetative form (*vegitante neoplastique*), but nevertheless is not truly fungating or cauliflower-like. Ulceration and necrosis with profuse bleeding were present in a marked degree in Case 1, and the gross appearance is best described as "geographical," i.e., firm elevations of tissue separated by sulci.

The uterine cavity could be explored easily by a large sound, the external os presenting as an irregular laceration formed by the intersection of three deep defects (Fig. 4). The extreme dark red "beefy" color of carcinoma was absent. The relative elasticity in the texture of the lesion is of value as a diagnostic sign. Although having an ulcerative lesion of advanced grade with crater formation at the time of examination, there was no history of bleeding and no blood was seen at examination (Case 2). The histological picture of tuberculosis is not always clear cut and typical tubercle formation is sometimes difficult to demonstrate in the picture of chronic infiltrative inflammatory disease.

TREATMENT

The form of the treatment depends upon whether the cervical lesion is primary or secondary. If the lesion can be definitely proved primary, surgery is indicated. We believe that panhysterectomy by the abdominal route is the best procedure, even if only a slight lesion is present, for by this method we are able to examine the pelvic viscera more closely for tuberculous lesions. If the vagina is involved its extirpation may be necessary (Jellett). Amputation of the cervix is advised perhaps only as a palliative measure in those cases in which for any reason more radical surgery is contra-indicated.

If the lesion is secondary to advanced tuberculosis elsewhere only palliative measures are indicated. Cauterization may give some relief from discharge. Astringent douches may be helpful, as tannic acid and zinc sulphate. Radium therapy may be used to some advantage but is absolutely contra-indicated if salpingitis is present (Jellett, Norris).

The general care of the patient is very important. It is advisable to treat a primary case promptly, since in any tuberculous patient the resistance is lowered to acid-fast organisms, and generalized systemic tuberculosis may follow.

The prognosis is good in primary cases. In secondary cases the prognosis depends upon the severity of the general infection. If the original lesion can be improved by general measures, the secondary infection will likewise have a greater chance of improvement.

SUMMARY

Tuberculosis of the cervix uteri is an extremely infrequent gynecological lesion. Less than 20 cases of undoubtedly primary tuberculous lesions in the cervix have been recorded. Secondary involvement of the cervix is much more frequent, and the prognosis is less favorable, depending upon the severity of the associated tuberculous foci. Two cases are presented, one of pantuberculosis of the pelvic viscera and a second a presumable case of primary tuberculosis of the cervix based on the evidence so far obtained. Complete recovery of the patient followed panhysterectomy in the first case.

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OSTEOPLASTIC RESECTION OF COSTAL ARCH FOR GUNSHOT WOUND OF SPLEEN[1]

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A VERY important principle of surgery is the development of a simple and readily accessible operative field.

During the night of May 8, 1927, I had to perform a splenectomy for an uncontrollable hæmorrhage from a gunshot wound of the posterior border of the spleen.[2] In order to gain proper access I was forced to do an osteoplastic[3] resection of the left costal arch. This procedure afforded such excellent opportunity to do careful surgery up under the vault of the diaphragm, in the presence of active bleeding, that I decided to report the case in order to bring the principle again to the attention of other surgeons.

In 1906 my father, Dr. Willy Meyer (3), published an article on the "Osteoplastic Resection of the Costal Arch in Order to Reach the Vault of the Diaphragm." He reported two cases in which he had performed osteoplastic resection of the costal arch. One was a case of an impermeable stricture of the lowest end of the œsophagus in a boy 14 years old. An attempt was made to reach the strictured area. A year later, 1905, he performed a splenectomy for a large sarcoma of the spleen in which the same technique was employed.

In 1924 I had the good fortune and opportunity to assist my father in performing one of these operations. It was for an inoperable carcinoma of the pylorus with total obstruction. In order to make an anterior gastro-enterostomy with Murphy button anastomosis near the cardia it was necessary to turn up the costal arch. Access was so perfect and convalescence so smooth that I was impressed with the great value of the procedure.

G. Marwedel (2) in 1903 was the first to publish an article describing the osteoplastic resection of the costal arch. Von Mickulicz has previously performed an operation on the same principle to gain better access, but the technique was much more complicated than that in the procedure of Marwedel. Following Marwedel's publication Asthoewer (1) wrote an article in which he stated that he had used a similar technique a number of times, the earliest instances being in 1894. Wiener (4) reported a case operated upon in 1908 in which he did an osteoplastic resection of the costal arch in a case of carcinoma of the cardia.

The author's patient was admitted to The Lenox Hill Hospital at 1:45 a.m. He was a well developed, muscular man who had been shot 5 times, by gangsters. Two bullets hit him at close range while he was lying on the floor of a hallway, after he had fallen from the first shots, and then three more bullets were fired into his body, hitting him in the left lower chest and in the back.

Examination revealed the following: The patient was in a state of shock. The left arm was numb, with partial loss of motion. The left clavicle was fractured at the junction of the middle and outer thirds. There was a bullet entrance wound along the anterior border of the left trapezius in the supraclavicular fossa. A second wound was present over the outer end of the clavicle. This had probably fractured the clavicle and then was deflected downward into the left pectoral region where the bullet could be seen and felt in a wound of emergence in this location. A third entrance wound was present in the anterior axillary line in the ninth intercostal interspace. The left side of the abdomen was held rigid and was tender. The left lumbar gutter was flat on percussion. Over the left twelfth rib posteriorly a bullet could be felt lying transversely and subcutaneously. In the back were two more entrance wounds showing that the direction of the bullets was downward. One was an entrance wound 2 inches from the mid-line on the left side in the tenth intercostal space, the other 2 inches from the mid-line on the right side in the lumbar region. There were no chest signs.

Laboratory examinations revealed: leucocytes, 27,600 with 86 per cent polymorphonuclears. No blood in the urine. X-ray examination showed one bullet in the left cervical region lying in front of the transverse process of the seventh cervical vertebra on the first rib, close to its articulation with the vertebra (Fig. 1). There was a comminuted fracture of the outer portion of the clavicle, and one bullet was lying in the soft parts of the left pectoral region. A third bullet was found close to a comminuted fracture of the twelfth rib on the left side. There was evidence of a bullet having passed through the twelfth rib (Figs. 2 and 3). The two other bullets were localized in the soft parts in the sacral and gluteal regions by a lateral X-ray view.

During the examinations the rigidity and tenderness of the abdomen had increased. The left lumbar region had become absolutely flat, and the temperature had risen to 101 degrees, the pulse to 110. A diagnosis of an acute abdominal condition was made. There was either a perforation of the splenic flexure of the colon or a wound of the spleen, the injury having resulted from a bullet which had entered the ninth intercostal space in the anterior axillary line, had then passed through the complementary space, and then through the diaphragm and either the spleen or the colon. Then the bullet must have passed through the twelfth rib and having lost its speed come to lie subcutaneously in this region where it could be palpated.

[1] From the Surgical Service of Dr. Carl Eggers at The Lenox Hill Hospital.

[2] This case was reported before the Surgical Section of the New York Academy of Medicine.

[3] The usual term applied to this operation is osteoplastic. The more correct anatomical term should be chrondroplastic, as the operation is on the cartilaginous, not the bony, portion of the ribs.

Fig. 1. Roentgenogram showing one bullet lying on the first rib in front of transverse process of the seventh cervical vertebra, and second bullet in the soft parts of the left pectoral region, with fracture of the left clavicle.

Fig. 2. Roentgenogram showing bullet in left lumbar region. This could be palpated subcutaneously. Also fracture of the twelfth rib with two other bullets in the soft parts of the sacral and gluteal regions.

Immediate exploratory laparotomy was indicated with a provisional diagnosis of rupture of the spleen.

A left pararectal incision about 4 inches long was made close to the mid-line. The peritoneal cavity was entered. No free fluid was present. When the left lumbar gutter and subdiaphragmatic space were explored a large amount of blood escaped. The spleen was palpated and was found to be small in size, high up under the vault of the diaphragm. A large tear was felt along the posterior border. Therefore, a transverse incision was made at right angles to the previous incision outward to the tip of the eleventh rib. With strong retraction on the costal margin it was found impossible to visualize the splenic hilus clearly on account of its high position under the vault of the diaphragm and the presence of a very much distended stomach (the patient had been eating and drinking heavily all evening). It was found that when the spleen was pulled downward and mesially, bleeding from the tear ceased, but when it was relaxed it bled freely again. Proper suture or tamponade of the tear on account of its inaccessibility was impossible, the tear being along the posterior border. Therefore splenectomy was considered the wisest and safest procedure. However, access to the hilus of the small spleen, which could not be sufficiently displaced downward, was very poor. Osteoplastic resection of the costal arch offered an outlook of material aid to obtain a proper operative field.

Fig. 3. More detailed roentgenogram of the fracture of the twelfth rib with the bullet and fragments of lead in the bullet tract.

OSTEOPLASTIC RESECTION OF THE COSTAL ARCH

The incision is lengthened upward to the level of the junction of the costal arch with the manubrium (Fig. 4). The line of cleavage between the posterior surface of the rectus muscle and its

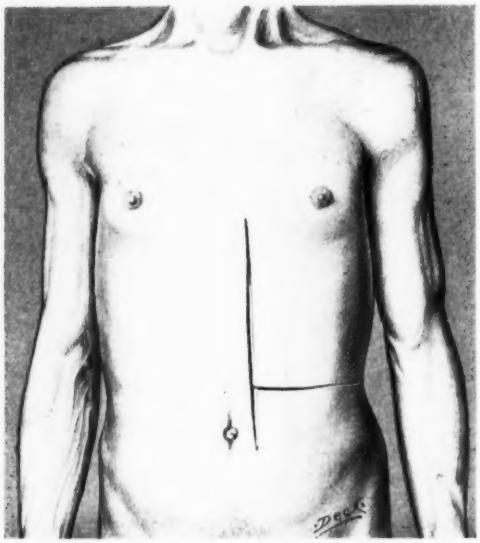

Fig. 4. Diagrammatic sketch showing abdominal incision lengthened upward to level of junction of costal arch with sternum and right angle transverse incision outward to tip of eleventh rib.

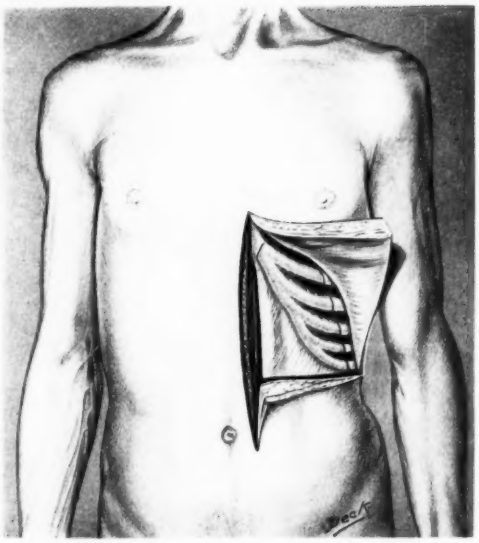

Fig. 5. Diagrammatic sketch showing exposed costal arch with division of the joined cartilages at the manubrium and the outer ends of the rib cartilages of the seventh to tenth ribs. Skin and muscle flap reflected upward and outward.

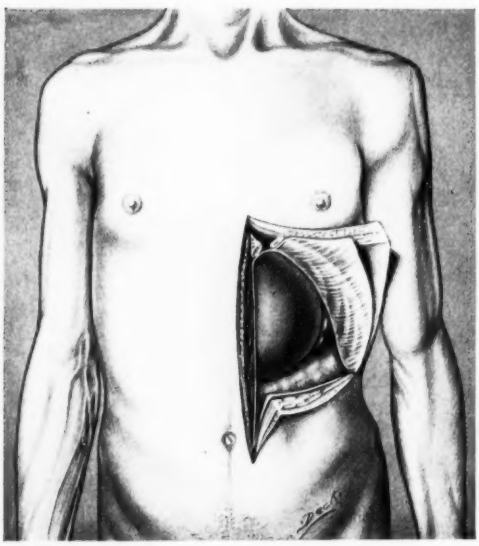

Fig. 6. Diagrammatic sketch showing skin and muscle flap and costal arch with peritoneum turned up giving excellent exposure of the organs lying in the vault of the diaphragm.

posterior sheath is found. This skin-muscle flap is then gently pushed upward and outward thus exposing the entire costal arch with the junction of the cartilages and the bony ribs.

With a sharp scalpel and great care not to injure the underlying pleura, the joined cartilage of the seventh, eighth and ninth ribs is completely divided near the manubrium. Care must be taken not to injure the internal mammary vessels, which lie just beneath. Then the outer ends of the seventh to tenth costal cartilages are also carefully completely divided close to the bony ribs. Sometimes it is necessary to divide a cartilaginous connection between the sixth and seventh cartilages. The procedure thus mobilizes the costal arch (Fig. 5). The skin-muscle flap is then turned back and the entire resulting skin-muscle-costal arch and peritoneal flap is turned up and retracted outward by the assistant's hand (Fig. 6).

Immediately, perfect exposure of the entire under surface of the diaphragm, the cardia of the stomach, and the spleen is obtained.

The actual splenectomy is now exceedingly safe and simple. The phrenico-lienal ligament can easily be ligated and divided, as well as an accessory vessel which runs to the lower pole of the spleen. The gastro-splenic vessels are easily

Fig. 7. Photograph of the spleen showing tear along the posterior border caused by the bullet.

divided and the main pedicle clamped, divided, and doubly ligated.

After the spleen had been removed in our patient the entrance wound of the bullet through the diaphragm could be seen as well as the wound of exit at the level of the twelfth rib posteriorly (Fig. 7). On account of the potential infection of the subphrenic space by the bullet, drains were inserted. The costal arch was then turned down again and a most careful suture of the abdominal wall was performed with interrupted chromic catgut in layers: peritoneum, muscle, and aponeurosis. The superficial wound was sutured with silkworm-gut and interrupted silk. A firm adhesive strapping dressing was applied.

Convalescence was stormy. The patient was cyanotic on the second postoperative day and bronchovesicular breathing could be heard, especially at the left base posteriorly. Bedside X-ray examination showed a small pneumothorax. Temperature was 103 degrees. On the seventh postoperative day the wound looked clean, and the skin sutures were removed. The pneumothorax had abated, but loud bronchial breathing could be heard over the left base. Physical examination revealed no pleural effusion. The patient coughed severely. A moderate seropurulent discharge issued from the drainage tract of the abdominal wound. On the eighth day after operation patient had a coughing spell which caused the entire perrectal portion of the wound to break open and the stomach and omentum to extrude. Cultures were made from the wound and staphylococcus albus was found in pure culture (probably carried in by the bullet). The viscera were replaced while the patient remained in bed and the wound edges were approximated with wide adhesive straps. Three weeks after the operation the temperature again began to rise and evidence of pleural exudation in the axillary line was found. Fifty cubic centimeters of slightly turbid fluid was withdrawn with the Potain aspirating apparatus, and cultures were reported to be sterile.

After 3 additional weeks with 10 days of intervening normal temperature, the bullet from the cervical region was removed. The bullet was found in an encapsulated abscess cavity within the scalenus medius muscle. The pus from this abscess also showed staphylococcus albus in pure culture as did the pus from the abdominal wound. After this bullet was removed, all the neurological findings of the left arm as found by Dr. Russel MacRobert quickly improved—local area of anæsthesia in the left forearm and hand of radicular distribution with motor disability which corresponded to the first thoracic segmental supply.

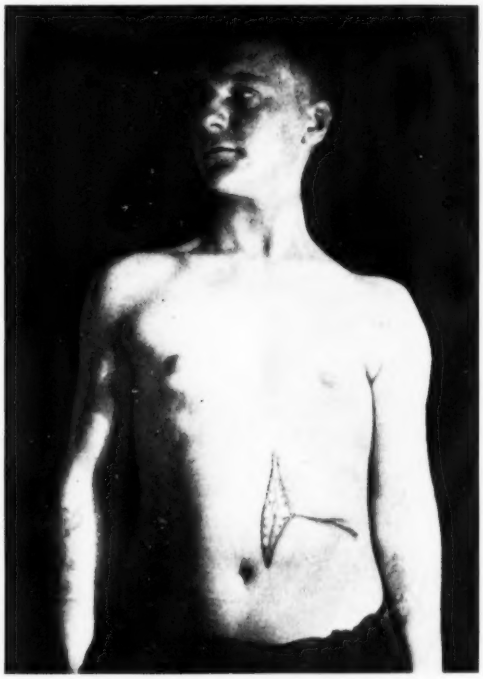

Fig. 8. Photograph of the patient taken 6 months after the operation showing healed scar with firm union of the costal arch.

Two weeks later the three remaining bullets were removed from the back, sacral and gluteal regions, under local anæsthesia. The bullet in the pectoral region, lying just in the wound of emergence, was removed at the time of the original operation.

The patient was discharged on the forty-third day with all the wounds healed.

At present there is evidence of weakness of the vertical portion of the scar where the wound had broken open. No actual hernia has occurred, but the patient is wearing an abdominal supporter. The costal arch has firmly united with the ribs. The pain, anæsthesia, and mobility of the left arm have entirely improved (Fig. 8).

CONCLUSIONS

Osteoplastic, or more properly called chondroplastic, resection of the costal arch is a great help in obtaining a good, simple, safe operative field in certain operations under the vault of the

diaphragm, in diaphragmatic hernia, and in operations on the liver, the spleen, and the stomach near the cardia.

The procedure is simple, quickly performed, and safe if done with the usual care always necessary in surgery.

The attachments of the diaphragm are not disturbed or interfered with.

The procedure justified itself as it affords excellent access to the subdiaphragmatic space. The final cosmetic and functional result is good and union of the costal arch is firm. The author begs to bring the procedure to the earnest attention of other surgeons.

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FOUR SPLINTS OF VALUE IN THE TREATMENT OF DISABILITIES OF THE HAND

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FOR many years Kanavel has emphasized the importance of properly designed splints[1] and of physical therapy for the prevention and correction of contractures following infections and injuries of the hand. Two principles have been particularly emphasized: the maintenance of the hand in the position of function during the period of forced immobilization, and the use of elastic tension to produce constant and painless traction[2] on fibrosed soft tissues and contracted joint capsules. When one observes how quickly the relaxation and mobility of contracted fingers and joints gained by a half hour or an hour of physical therapy is lost because of the lack of any form of retentive apparatus, he is doubly impressed with the importance of careful splinting in the treatment of contractures.

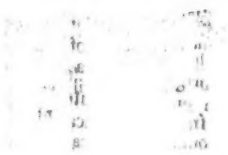
Four splints have gradually been developed in our work which have proved of great value in the carrying out of the principles mentioned. All are light in weight, fairly rigid, and made of hard, flat aluminum, No. 2SH, .081 inch in thickness. The first (Fig. 1, a, b) is designed to maintain the hand in the position of function, i.e., with the wrist dorsiflexed, the thumb abducted from the hand and facing the fingers, and the fingers semiflexed, as though grasping a tennis ball. The splint is curved in its long axis so as to fit snugly to the volar surface of the forearm; it is slightly curved in a transverse axis just distal to the wrist so as to fit the "heel" of the hand. Pressure on the thenar eminence is eliminated by the cutting out of a rounded portion on the radial side. Dorsal flexion is regulated by the degree of flexion of the splint at the wrist. The lower, rounded end of the splint separates the thumb from the fingers, but stops short of the metacarpophalangeal articulations so as to permit flexion of the fingers at these joints, the joints most often held immobilized after infection and injury, and as a result, most often involved in fibrous contractures.

Such a splint can be padded with washable "feather edge" rubber, or with felt (Fig. 1, c, d); it can be applied to the hand while infection is still present, as soon as it is possible to substitute intermittent hot, wet dressings for a continuous, wet dressing—usually within 4 or 5 days of the onset of even a serious infection. If padded straps with buckles are attached to the splint, it can be quickly taken off and reapplied—important factors if one is caring for a considerable number of patients or is treating patients who are not particularly interested in getting well quickly and therefore tend to neglect the treatment indicated.

The second splint (Fig. 2) is similar to the first, with the addition of a raised aluminum crosspiece

[1] For some years because of our difficulty in finding anyone to manufacture the splints described in exact accordance with our ideas and at what seemed to us a reasonable cost they were made by Dr. Michael L. Mason and myself in the carpenter shop of the hospital. Lately, we have succeeded in having them made for us according to our exact specifications by the American Metal Ware Co., 368 W. Huron St., Chicago.

[2] Unless traction is painless it cannot be long continued nor without injury. Scar tissue can be stretched with impunity if moderate traction is maintained for long periods; if it is forcibly broken down hæmorrhage takes place, more scar tissue forms, and the ultimate condition is worse than the first.



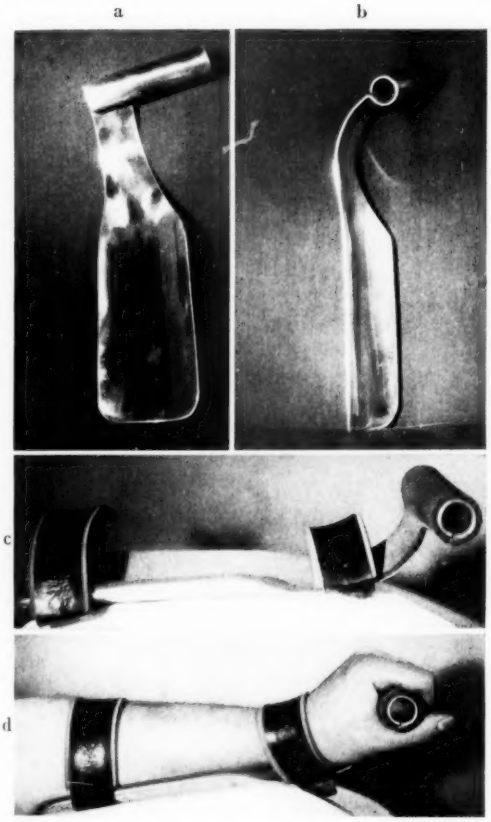


Fig. 1. Aluminum splint for maintaining the hand in the position of function.

at its upper end (Fig. 2, a) to which straps with buckles, steel springs, and leather loops can be attached for the production of elastic tension on the thumb and fingers. Figure 2, b shows the splint with straps for each finger. A sixth strap may be attached to the loop over the thumb, to pull directly upward, (in the line of the radius), and thus help to pull the thumb away from the hand into the abducted position, a consideration of particular importance if the thumb has been allowed to lie for days or weeks alongside the hand in the extended and prone position.

The elbow cuff (Fig. 2, a, c) attached by straps and buckles to the upper corners of the splint helps to keep it from slipping down past the metacarpophalangeal joints when the pull on the fingers is increased.

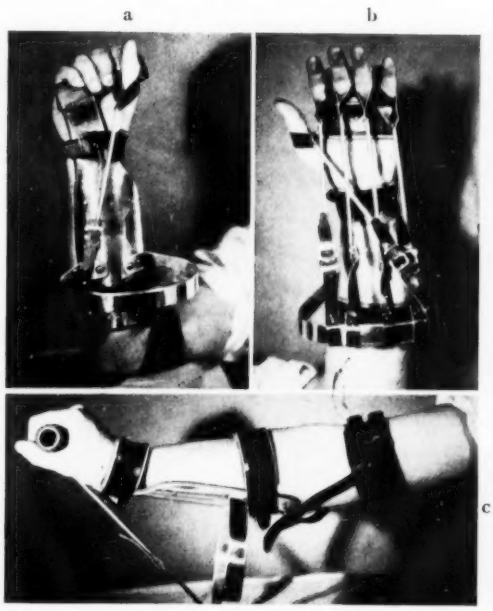
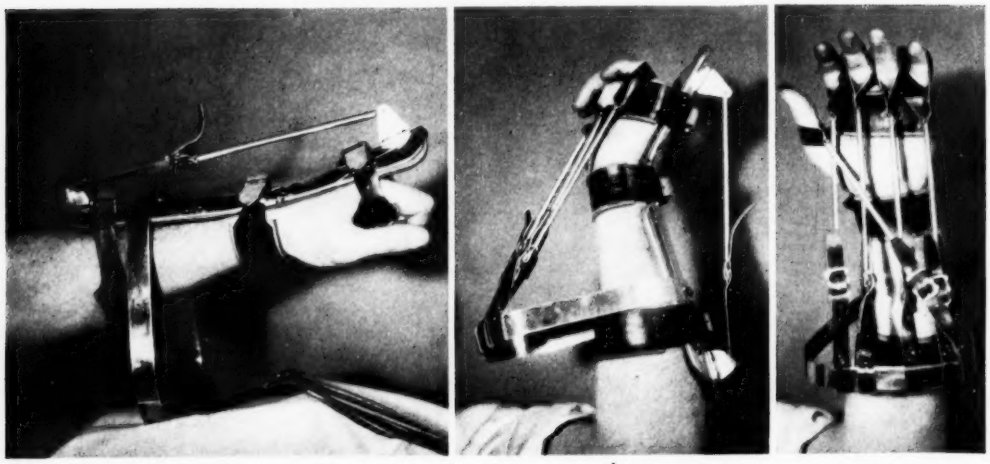


Fig. 2. The same splint as in Figure 1 with cross bar, straps, buckles, springs, and loops for the production of elastic tension on the fingers and thumb, so as to flex the fingers and abduct the thumb from the hand. (In a and c only one strap, buckle, spring, and loop are shown so as not to make the illustration confusing from a multiplicity of straps.)

The third splint (Fig. 3) is designed to produce the same effect as the second, but is applied to the dorsum of the forearm and hand in cases in which the presence of wounds or operative incisions makes it desirable to avoid any pressure on the volar surface, or in cases in which involvement of the wrist joint makes it desirable to bring elastic tension to bear on the periarticular structures of the wrist. Dorsal flexion at the wrist is obtained with the aid of a hinge at the wrist, and a backward pull on the hand secured with the aid of a strong spring on the back of the splint. The effect produced on the fingers and thumb is exactly the same as with the second splint. The elbow cuff to prevent the splint from slipping distalward, shown in Figure 2, is attached in the same fashion to splint 3, but has been omitted in the illustration shown. Pressure on the styloid process of the ulua is eliminated by cutting out a rounded portion of the splint on the ulnar side just above the hinge.

The fourth splint (Fig. 4), like the third, is



a b c

Fig. 3. A splint designed to produce the same effect as splint 2, but to be applied to the dorsum of the hand.

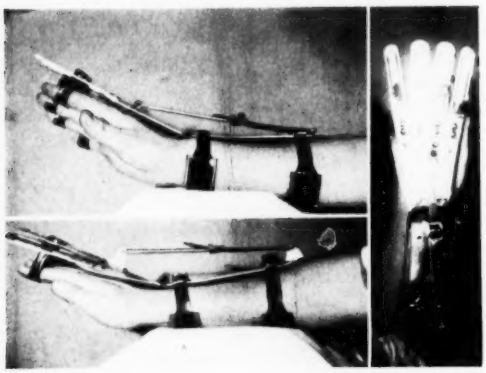


Fig. 4. A splint to produce extension of flexed fingers and wrist with the aid of elastic tension.

hinged at the wrist and is applied to the dorsum of the forearm and hand. It is designed, however, to aid in the extension of contracted fingers, and not, as splints 2 and 3, in the flexing of them. Slotted extension pieces for each finger and for the thumb, if desired, are riveted to the hand piece, and tension of any desired degree is applied to the flexed fingers with the aid of rubber or elastic loops and buckles. This splint is helpful in the treatment of tendon contractures, and particularly in the treatment of that type known as von Volkmann's contracture. In the latter condition it is frequently impossible to extend the sharply flexed fingers until volar flexion at the wrist relaxes the contracted tendons. The hinge at the wrist in the splint illustrated permits volar flexion, of any desired degree, at the wrist. As the contracted tendons are gradually stretched, one is able to straighten out the wrist and maintain tension of any desired degree by tightening the strap on the back of the splint, and, if necessary, increasing the strength of the springs or adding a second spring.

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SPONTANEOUS RUPTURE OF PYOSALPINX INTO THE URINARY BLADDER

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WHILE we present in this article an instance of rupture of pyosalpinx into the urinary bladder, it must be noted that pelvic affections involving the female internal genital organs or intestines, whether of inflammatory or neoplastic origin, will occasionally create a fistulous communication with a neighboring viscus, which results in a spontaneous evacuation of accumulated pus. Rather infrequently such processes have involved the bladder alone and ruptured into its cavity. However, cases have been reported in which occasional rupture has occurred into the urinary bladder as a result of appendicitis; infected dermoids; abscesses of the ovary; extra-uterine pregnancy; pelvic tuberculosis; tubal diseases of parasitic origin; malignant diseases of the uterus, adnexa, and intestines; and postabortal pelvic infection. The case of Fronstein and Serdjukoff, in which an ovarian abscess ruptured into the bladder, and the one of Sbrozzi, in which, in the course of an active pyosalpingeal process, the adherent intestine opened into that viscus, are conspicuous oddities illustrating bladder fistulæ.

Spontaneous rupture of tubal abscesses into the bladder occurs rather infrequently. Under the broad classification of pelvic abscess, Perrimond in 1897 collected 67 cases showing clinical evidences of spontaneous rupture into the bladder. In only one instance could it be definitely stated that the communication was limited to a tubal pus cavity. The other cases are described as being pelvic abscesses or abscesses of the broad ligament with varied spontaneous ruptures into the bladder, vagina, intestines, and rectum, or through the skin. A number of his cases followed parturition or abortion, several were infected dermoids or other cysts of the ovary, a few were infected ectopics. In the light of present day methods of diagnosis, aided by cystoscopy, all his cases appear to be poorly elucidated and cannot, with the exception of one case, be included in our series. Similarly, Delbet has collected 958 cases of pelvic suppuration, most of them without reported cystoscopic or operative findings. Of his cases of alleged rupture into the bladder, none could be included in our series because convincing proof is lacking.

Spontaneous rupture is an unusual accident resulting from a distention of a pyosalpinx which is often secondarily infected. Though a number of these cases are primarily due to the gonococcus, many of them are of tuberculous origin. As usual, under such conditions the pus tubes are enlarged and their close proximity to a hypersensitive and overdistended bladder results in involvement of these structures, and a firm union occurs at some point as a result of a plastic exudate. Continuous pressure at the contiguous area causes the formation of a weak spot in the bladder wall and results in gradual and subsequent sloughing and rupture.

SYMPTOMS

The patients suffer from the usual symptoms of adnexal disease, either of the acute or chronic type. Occasionally, the initial attack is severe enough to involve the bladder and produce rupture, following which there is a sudden abatement of all symptoms, but often the original condition recurs subsequent to a temporary closure of the fistula, the whole train of symptoms reappears, and the patient slowly lapses into a condition of chronic invalidism. One case (Muller and Petitjean) was studied for 10 years before its true nature was discovered. Along with the symptoms of pelvic disease, there usually are prodromal symptoms of bladder disturbances, namely, discomfort, frequency, tenesmus, cloudy urine, and occasionally hæmaturia. During this stage the pelvic pain and discomfort are aggravated, the general condition is decidedly worse, chills and rising temperature supervene, and when rupture occurs there is often an attack of sharp pelvic pain and a sudden appearance in the urine of a large quantity of frank, foul-smelling pus. As already stated, this marked pyuria is followed by a decided drop in the temperature and an abatement of all symptoms. Examination at this time reveals a diminution in the size of the offending mass. In a small number of cases, there are no prodromal bladder symptoms and the sudden pyuria gives the first indication as to what is occurring. The maximum pus content is usually at the onset, i.e., at the time of the first rupture. A thick, greenish or yellowish, foul-smelling pus is characteristic.

The course after rupture is variable. The pyuria may last a few hours or a few days and then clear up entirely. This is a period of apparent cessation, during which time the pelvic

condition remains quiescent. However, the pathological process from another tubal exacerbation may cause renewal of symptoms and a re-appearance of the pyuria consequent upon the re-opening of the fistula. The patient's general condition thus becomes progressively worse; most of the reported cases were in distressingly poor condition when they presented themselves for final care. The instance of a 10-year duration has already been alluded to. This chronicity and invalidism are observed principally in the tuberculous cases. Frequently, in fistulæ due to a pyosalpinx of gonococcal origin, a spontaneous cure of the fistula occurs.

DIAGNOSIS

A clinical condition so characteristically manifested as just outlined is not likely to escape one's notice. A reasonable amount of alertness will often establish it from the train of symptoms. Yet, the absolute aid afforded by the cystoscope makes it possible to make the diagnosis by this means alone. The finding of an opening in the bladder and skiagraphy of a contrast medium after filling a neighboring cavity through a ureteral catheter is all that is necessary to determine the existing pathological condition.

CYSTOSCOPY

The finding of an opening in the bladder wall and cystography of the neighboring cavity after it has been filled with a contrast medium, such as an iodide solution, iodopin, or lipiodol, will firmly establish a diagnosis already presumptive on clinical evidence. Sometimes only an isolated area of redness in an otherwise normal bladder is seen at the site of expected rupture, while on subsequent examination a characteristic puckering and possibly a crater-like opening may be present. A normal looking bladder wall, except for the tell-tale isolated area just described, is often found. Characteristic indeed is the presence in this reddened, œdematous, puckered area of a small opening through which pus is seen exuding and into which a small catheter can be passed, but investigation does not always reveal this condition.

The use of three catheters, one into either ureter and the third into the pus cavity, further clarifies the situation. This method was used in the case recorded by Beer.

In the collected cases it is often reported that cystoscopy performed some time after rupture, with clear urine, showed no opening, but only the tell-tale reddened and puckered area in sharp contrast to an otherwise normal mucosa. As this condition was evident in the case presented, we resorted to another method of proof not previously attempted, but yet confirmatory.

A long, large bore, aspirating needle was plunged into the offending adnexal mass through the vault of the vagina and a quantity of thick, foul-smelling pus was withdrawn (the pus had the same physical and cultural characteristics as that previously noted in the urine). With the needle remaining *in situ*, a quantity of a 20 per cent solution of sodium iodide was injected into the cavity, the needle was then withdrawn, and the vagina tamponaded. The bladder was catheterized and a skiagram immediately taken (Figs. 1 and 2). The pus cavity was clearly outlined, and, in addition, a small quantity of the opaque substance was found in the bladder. The bladder accumulation was then drawn off and was found to contain iodine. Subsequent hourly examinations were continued throughout the day and each of these revealed the presence of iodine. The usual site of rupture occurs on either side of the ureteral openings on the lateral or posterior wall.

TREATMENT

The case of Duvergey and Dax, considered by them as being the first case (1922) in which salpingography was used as an aid in the diagnosis of this condition, was treated by transvesical intratubal instillation of 1 per cent silver nitrate. Their comment is that the bladder symptoms cleared up and the adnexa became reduced in size. Other cases have responded to bladder lavage, rest in bed, and supportive measures, while others have done well after simple colpotomy. It is reasonable to suppose that frequent evacuation of the pus cavity, combined with rest and various other measures, will greatly help to restore a patient to a fair degree of health. Most of the patients are in such poor general condition when first seen that one is never anxious to assume an operative risk until after the general health has been improved.

A good method of treating these cases is as follows: When rupture takes place, bladder lavage and urotropin or hexylresorcinol should be given until the urine clears and vesical symptoms subside. Transvesical lavage of the pus cavity is valuable, as it favors drainage. If pelvic abscess intervenes, colpotomy can be done. If the inflammatory process thus comes under control, the best time to operate is after a short period of levelled temperature and a repeated white cell count of 10,000 or less. The operation by laparotomy should aim to remove all pathological tissue. The site of rupture into the bladder

Fig. 1. Pyosalpingeal abscess. A large trocar needle has been inserted into the cul-de-sac.

Fig. 2. The pyosalpingeal abscess is filled with a solution of 20 per cent sodium iodide. The abscess cavity is well outlined and a communication between the abscess cavity and the bladder is visible. Some of the iodide solution is seen in the bladder.

should be sought out and the damage repaired either by simple suture or by resection of the ulcerated area. In our opinion these operations are not complete unless drainage through the vault of the vagina is effected in the cases due to a non-tuberculous pyosalpinx.

PROGNOSIS

Of the 34 patients reported, 9 were not operated upon; of these, 1 died, 2 reported that their health was improved, and 6 reported cured or end-result not known. Of the 25 patients who were operated upon, 14 were cured, 1 responded to transvesical instillations (Cotillon), 2 were not reported, and 8 died. The ages ranged from 22 to 48 years. The presence of tubercle bacilli was recorded 6 times, of tubercle and colon bacilli once, and of streptococci and colon bacilli once. In 1 case no growth resulted, while in 23 cases the type of organism found was not reported.

CASE REPORT

W. D., a widow 35 years of age, was admitted to the Harlem Hospital on March 24, 1927, with a proved gonorrhœal urethritis and endocervicitis of 3 weeks' duration. During that time she had had severe pains in the lower abdomen, chilly feelings, and frequent and painful urination. Pelvic examination revealed the presence of bilateral tender masses. The left mass was larger than the right and bulged anteriorly into the vault of the vagina and likewise into the cul-de-sac. The first urinalysis showed many pus cells. The temperature ranged from 101 to 103 degrees, but slowly subsided. Cystoscopy on March 28 revealed a congested mucosa and on the left wall of the bladder beyond the ureteral openings a much darker area but no puckering.

One week after admission, following a sudden severe pain in the lower abdomen, the urine became thickened with a greenish-yellow, foul-smelling pus. There was prompt relief of the pelvic pain and an improvement in the general condition. The left lateral mass was also reduced in size. Thus far, the clinical manifestations indicated that the left lateral mass had ruptured into the bladder. When the urine began to clear, about 5 days after the rupture, a more extended examination was undertaken.

A catheterized specimen yielded the same thick pus just described and, on cystoscopy, at the site on the left lateral wall of the bladder, was an area of œdema, infiltration, and plication of the mucosa, covered with mucus and placques of epithelial débris. The rest of the mucosa was only slightly congested. No direct opening was found, and no new pus contaminated the field during the examination. Smears and cultures of the pus showed cocci in twos and in short chains.

At this time an aspirating needle was plunged through the vagina into the left lateral mass (Fig. 1). A quantity of pus was withdrawn and was found to be similar in all respects to that found in the bladder. Through the needle, left *in situ*, a quantity of 20 per cent sodium iodide solution was injected and a roentgenogram taken. The opaque fluid was seen in the pus tube region (Fig. 2) and some of it was also seen in the bladder. A catheterized specimen at this time and other specimens obtained during the day revealed the presence of the iodide. After subsidence of all symptoms and with levelled temperature and satisfactory blood count (under 10,000 white blood cells), an operation was performed by one of us (di P.) on April 15. Bilateral adnexal suppuration was encountered and properly dealt with and in the presence of a few intramural fibroids hysteroplasty was performed. The site of the bladder communication was found and Lambertized and drainage established through the cul-de-sac with iodoform gauze. The convalescence was uneventful and the patient left the hospital on May 22, 1927.

CONCLUSIONS

1. Spontaneous rupture of a pyosalpinx into the urinary bladder is very rare.

2. The symptoms are clinically characteristic. Following sharp pelvic or suprapubic pains, large quantities of frank, ofttimes foul-smelling pus appear in the urine and there is a drop in temperature, a relief of urinary and vesical symptoms, and a decided improvement in the general condition of the patient. Cystography and cystoscopy add convincing data to an otherwise obvious clinical diagnosis.

3. A patent ostium is not always visible in the bladder wall, but an isolated area of œdema or redness with a crater-like central depression, together with pelvic pathology, is presumptive evidence of impending ulceration or rupture. The site of the rupture is usually on the lateral wall, just beyond and to one side of the ureteral opening. An absolute diagnosis is reached by means of roentgenograms taken after the pus cavity has been filled with opaque fluid, either transvesically or by the authors' method.

4. A rupture may heal spontaneously, as may also the original abscess, and later recur several times, especially if the patient is in poor general condition as a result of chronic invalidism.

5. Operation by laparotomy to remove all disease is the proper method of treatment and is best undertaken when the temperature is levelled and the white cell count less than 10,000.

6. The fistulous communication between the pus sac and the bladder cannot always be found at the time of operation, but when it is found, it is given the necessary surgical attention. Drainage by indwelling catheters and by gauze through the vaginal vault is recommended.

CASES REPORTED IN THE LITERATURE

The following 34 cases, including the authors', were gathered from the literature. As previously stated, many reports could not be included in this study because of the lack of sufficient evidence.

1. Zurhelle's case. The patient was 35 years old and had had a purulent urethral discharge for 4 months. The urine showed bacillus coli in great numbers. Cystoscopy showed that the bladder was normal except for a tuft of whitish tissue near the right ureteral ostium into which pus could be expressed by external pressure. Operation revealed bilateral pyosalpinx, which was treated by marsupialization of the sac and the insertion of an indwelling catheter. The patient was discharged cured in 7 weeks.

2. Carlier's case, reported by Fauverghe. The patient, 44 years of age, had had pelvic pains for 3 years. Rupture into the bladder occurred after several days of vesical symptoms and was followed by marked improvement in the general condition. The end-results are not known.

3. Reymond's case, reported by Fauverghe. A patient 24 years old had previously had pus in the urine but no cystitis. Four years before coming under observation, she had had symptoms of cystitis for 5 weeks preceding rupture. A large quantity of pus and blood was present at the time of rupture and this condition persisted for 4 years. Cystoscopy showed the bladder wall to be normal, with a fistulous opening on the right side. Pus flowed through the opening on outside pressure. Colon bacilli were demonstrated. The end-results were not reported.

4. Guyon's case, reported by Gras. The patient, 35 years of age, developed pelvic pains early in 1904, culminating in September in signs and symptoms of rupture into the bladder. Cystoscopy revealed on the right side of the bladder an isolated area with a central crater through which no pus was seen to exude. Several periods of remissions and exacerbations occurred after 3 months' observation. The end-results are not clearly indicated, although the patient evidently still had cystitis and cloudy urine in 1905.

5. Koenig's case, reported by Fauverghe. The only information given is that after sudden pain a tumor on the left side became smaller and pus appeared in the urine. The condition persisted for a period of 14 days. The end-result is not noted, but the case is reported as cured.

6. Case of Lauchlan-Aiken, reported by Fauverghe. A patient 30 years old had a tumor on the right side which became smaller with the appearance of pus in the urine. Cure resulted in 2 months.

7. Case of Panthel, reported by Fauverghe. A 36-year-old patient developed postpartum infection, followed by discharge of pus (perforation) by rectum. Seven days later, after severe pains, pus appeared in the urine. Death was due to severe sepsis.

8. Case of Chever, reported by Fauverghe. For 6 months the patient, 31 years of age, had had symptoms of strangury, and a large quantity of pus was noted in the urine. Final recovery is recorded.

9. Case of M. Polosson, reported by Perrimond. The patient, 25 years of age, developed postabortal infection. Examination revealed the presence of a tumor on the right side of the pelvis. The urine was foul smelling, turbid, and purulent. Colpotomy with bladder lavages resulted in cure after 1 month.

10. Case of Michaux, reported by Lafourcade. For 6 months the patient, 36 years of age, had had postabortal infection. Then malodorous pus suddenly appeared in the urine and was noted for 3 days. Vaginal hysterectomy was performed, but the patient died.

11. Case of Richelet, reported by Lafourcade. The patient, 28 years of age, had had symptoms for 2 years and pus had been noted in the urine at intervals. Vaginal hysterectomy was performed, and the patient's general condition was reported as good.

12. Case of Dubar, reported by Fauverghe. A left pyosalpinx ruptured into the bladder and vagina in a patient 38 years old. Vaginal hysterectomy was performed and complete recovery resulted after 1 year of pelvic pain and abdominal fistula.

13. Case of Dubar, reported by Fauverghe. A patient 27 years of age developed postoperative infection in 1901. Pains were felt in the pelvis for several weeks, but were relieved suddenly after the appearance of pus in the urine. Cystoscopy confirmed the diagnosis of perforation of the left pyosalpinx into the bladder. Operation consisted of removal of abscessed adnexa and bladder irrigations. Cure resulted in 1 month.

14. Case of Legueu, reported by Gras. The patient, 40 years old, presented a history of sudden severe pains, vesical symptoms, and pus in the urine 3 years before.

Fig. 3. Cystoscopic appearance just before the rupture occurred.

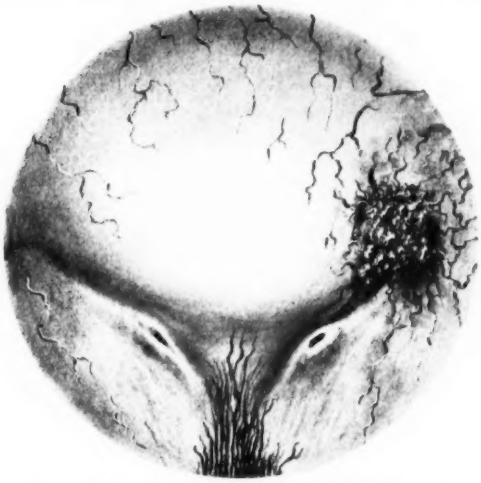

Fig. 4. Cystoscopic appearance several days after rupture has taken place. Note the absence of any aperture.

Cystoscopy revealed a normal bladder wall, but the right ureteral opening showed swelling. Beside it was seen a small opening through which pus escaped. Panhysterectomy was done. No communication with the bladder was found, although the right pus tube was found to be adherent to the bladder. Vaginal as well as abdominal drainage was instituted, but patient died.

15. Case of Muller and Petitjean. For 10 years a patient 38 years of age had had symptoms indicating perforation into the bladder of a purulent process. Bacteriological examination of the purulent urine and cystoscopy were negative. Hysterectomy and removal of adnexa were done and no communication with the bladder was found. No drainage was instituted. At autopsy the site of the perforation was discovered.

16. Case of M. Bulcke. The patient, 25 years old, had had purulent urine for 2 months. Cystoscopy revealed a fistula in the posterior wall and at operation it was found to communicate with a large left pyosalpinx. An indwelling catheter was inserted. Recovery.

17. Case of A. Funke. The patient had had pus in the urine for 3 months after parturition. A perforation was found on the left posterior wall. Laparotomy was performed. A remnant of the pus sac left adherent to the bladder wall obscured the perforation. Cure after 24 days.

18. Case of A. Funke. The patient, 33 years of age, reported that rupture into the rectum had occurred prior to rupture into the bladder. At operation a communication between the bladder and the right pus tube was found. Recovery occurred within a year.

19. Case of A. Funke. At laparotomy two perforations into the bladder were found and sutured. Recovery. Only one other case has been reported in which more than one fistula was found.

20. Case of A. Freund. Chronic suppuration into the pelvis had been noted for a few years following several operations for fistulous openings into the bladder and intestines and through the abdomen. The patient was in poor condition. At laparotomy, the abscessed adnexa were removed and the two perforations into the bladder were sutured. Death.

21. Gayet's first case (1922). The patient, 42 years old, had had pyuria and lumbar pains for a year and was very feeble as a result of the long illness. A mass was noted on the right side. Cystoscopy revealed some œdema of the upper and posterior wall of the bladder and exudation of pus over several spots in this zone. The rest of the bladder wall was normal. Operation was followed by cure. Guinea pig inoculation showed tuberculosis.

22. Gayet's second case (1922). The patient, 23 years of age, had had considerable pus in the urine. Cystoscopy showed false membranes in the bladder. The catheterized kidney urines were clear and negative on inoculation. A second cystoscopy showed a bulbous area above the left ureter, and in the center of this there was a slit filled with a plug of pus. Pressure on the vagina caused a discharge of pus through the opening. Operation revealed a left pyosalpinx, tuberculous in character. No opening into the bladder was found. Cure.

Gayet reports 3 additional cases of "tuberculous pyosalpinx opening into the bladder," but the openings were not clearly demonstrated.

23. Auvray's first case. The patient, 38 years of age, was in poor general condition. She was admitted to the hospital in March, 1912, and a diagnosis of right salpingitis was made. The urine contained pus, but no cystitis was present. Cystoscopy showed the right side of the bladder to be reddened, but no opening was seen. The condition improved after lavage. Cystoscopy one month later showed a characteristic opening and exuding pus. At operation, performed on July 2, 1912, a fistula was found and sutured. Cure.

24. Auvray's second case. The patient was admitted to the hospital on December 4, 1912. Pus had been noted in the urine for 2 months. Cystoscopy showed the anterior wall to be red and vascular. The left ureteral opening was gaping and surrounded by a red congested area. Just outside of this was seen a small area of œdema with a central depression, from which pus exuded. At operation the opening into the bladder was found and sutured. Death.

25. Auvray's third case. The patient was admitted in June, 1913, after 2 months of illness due to pelvic disease.

After several operations she left hospital only to return in June, 1914, because of the re-appearance of pelvic pains, pus in the urine, and vesical symptoms. A skin and bladder fistula was demonstrated. At operation, a right pus tube with a bladder opening was found and sutured. Death occurred 25 days later.

26. Cotillon. The patient, 24 years of age, had had symptoms of pelvic and pulmonary disease for 3 years. No urinary symptoms had been present. In October, 1919, after severe pelvic pain, painful urination, and cloudiness were present for 4 or 5 days. In January 1920, examination revealed pelvic masses, the larger one on the left side, purulent urine, and pus in excess on several occasions after pressure on the masses. The urine did not reveal the presence of tuberculosis, but guinea pig inoculation was positive. Pulmonary tuberculosis was present. A small opening into the bladder was found on cystoscopy. At operation, tuberculosis of the adnexa was discovered. Death.

27. Case of Duvergey and Dax. The patient, 34 years old, was admitted to the hospital in February, 1922. She complained of fatigue, malaise, and painful, frequent, cloudy urination. Suddenly the urine became thick and cloudy. Examination revealed a greatly emaciated patient with uterus and adnexa massed together. Cystoscopy showed a small opening on the right side in the center of radiating folds, but no visible discharge was noted through this opening, which was large enough to admit a catheter. The diagnosis was confirmed by means of roentgenograms made after an opaque fluid had been injected into the cavity, transvesically through the small cavity. This is probably the first recorded instance of salpingography. The symptoms improved after transvesical lavage with 1 per cent silver solution.

28. Davidson's case (1927). The patient, 29 years of age, had had pelvic pains and bladder symptoms for a long time and was very weak. The urine was purulent; bacillus coli communis was demonstrated. Cystoscopy showed marked œdema at the base, obscuring the ureteral orifices. At a subsequent examination the œdema had cleared sufficiently to show that the ostia were normal and the presence of a small puckered area internal to the left orifice. At operation an opening into the bladder was found and sutured. Cure.

29. Beer's case. In October, 1928, a patient 22 years old gave a history of painful micturition, especially about menstruation time, for 2½ years, and severe exacerbation 10 weeks before. The urine was cloudy, but no blood was present. Cystoscopy showed a normal bladder, but behind the trigone toward the left was an area of œdema. In the center of this area was a strawberry-like excrescence with a drop of pus at the summit. By the transvesical use of an opaque fluid, a tumor to left was found communicating with the bladder and also with the intestines. Tuberculosis was also present. Operation was followed by cure.

Interesting in this connection is Sbrozzi's case of vesico-intestinal fistula, due to pyosalpinx, but differing from Beer's case in that the pyosalpinx, although adherent to the bladder and intestine, did not itself connect with either. Still another interesting case, paralleling our cases, although dissimilar, was that of an ovarian abscess which ruptured into the bladder. This case was reported by Fronstein and Serdjukoff.

30. Joly's first case. In August, 1926, a patient 35 years of age reported that she had had salpingitis since 1924. A mass was present on the left side. Following sudden pains in the pelvis the urine became cloudy and fetid. Relief of symptoms and diminution in the size of the mass were noted. Cystoscopy showed the bladder and ureteral orifices to be normal. A granular protrusion surmounted by a pustule was seen above the left ureter. Operation: left salpingectomy and hysterectomy. The site of the bladder corrosion was found and sutured and an indwelling catheter inserted. Subsequent cystoscopy showed a scar at the site of the perforation. Cure.

31. Joly's second case. In September, 1926, a patient 32 years old was examined and a diagnosis of bilateral adnexal disease was made. Colpotomy was performed in order to evacuate the left abscess, which was larger than the right one. Following sudden painful micturition, a large quantity of pus was noted in the urine. The patient was still under observation at the time Joly made his report.

32. Violet and Chalier's first case. In 1905 a patient 37 years old was examined and found to have bilateral tubal and pulmonary tuberculosis. At operation, an opening into the bladder was found and sutured. Three years later the patient was reported as cured.

33. Violet and Chalier's second case. In 1906 a patient 48 years old was operated upon. A right ovarian tuberculous abscess and right pyosalpinx were found. There was also a left tubo-ovarian abscess (probably tuberculous) adherent to the thickened bladder. The cavity of the bladder contained 60 cubic centimeters of pus, similar to that in the left adnexa. No actual perforation was found. Cure.

These authors report a third case, also tuberculous, in which there was wide pelvic suppuration. This was not clearly a case of pyosalpinx and is therefore simply referred to and not added to our list.

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THE COINCIDENCE OF HYPERPLASIA ENDOMETRII AND CARCINOMA CORPORIS UTERI

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HYPERPLASIA of the endometrium is the most important single factor concerned with abnormal uterine bleeding at the time of the menopause. In this respect, it must constantly be considered in the differential diagnosis from carcinoma of the body of the uterus and it is thus of prime importance to determine its exact relation to malignancy and how frequently the two conditions may coexist.

There have been but few cases reported in which carcinomatous areas were found in hyperplastic endometrium. In 1906, Doca (3) described a specimen obtained from a patient 45 years of age. The uterus was involved in a diffuse myoma, the mucosa was markedly thickened and presented the usual appearance of hyperplasia of the endometrium, while in one area could be seen a definite early glandular carcinoma.

R. Schroeder (13) found definite malignant changes in the hyperplastic endometrium of 2 patients. Since these women had passed the menopause several years before, he was uncertain as to whether the condition should be regarded as a simple hyperplasia endometrii or as more in the nature of a diffuse adenoma with a tendency to malignant degeneration. In a more recent work (14), however, he mentions the possible occurrence of carcinoma in hyperplastic endometrium and presents an illustration of this phenomenon. Ewing (4) states that he has seen 3 cases of carcinoma arising in the hypertrophied glands overlying the most prominent points of submucous myomata. Finally, R. Meyer (10) has reported 5 very interesting cases of hyperplasia endometrii associated with malignancy, and he was able to point out a very important fact, namely, that an adenomatous cancer not only may have its origin in hyperplastic mucosa, but may arise directly from previously simple hyperplastic glands.

The use of repeated curettages in patients with hyperplasia of the endometrium has shown that this lesion is occasionally succeeded by a carcinomatous condition. Baecker (1) has described the case of a woman who was curetted 20 times over a period of 10 years. The first seventeen curettings showed an "endometritis glandularis," the next two an adenoma benignum, and finally an adenoma malignum. A somewhat similar experience was reported by Horsley (8), who found a "hypertrophic endometritis" on two occasions, while the third operation revealed a low grade adenocarcinoma.

In this connection, reference must also be made to a rare condition which was recently reviewed by one of us (Fluhmann, 6). In this lesion, unusual masses of epithelial cells, resembling the basal-layer cells of squamous epithelium, occurred in close association with the glands of hyperplastic endometrium. The exact significance of this change has not been determined with certainty and although there is every reason to believe that it represents a benign process in the nature of a metaplasia of cylindrical to squamous epithelium, a number of authors who first described it considered it as unquestionably carcinoma.

Although the term "precancerous lesion" has been applied to "endometritis glandularis" by a few authors (McCann, 9; Findley, 5), the evidence advanced is not conclusive and the consensus of opinion seems to be that the association of hyperplastic endometrium with malignancy is very unusual. It was apparently not noted in the cases of cancer of the corpus uteri studied by Schottlaender and Kermauner (12), and the only case described by Cullen (2) in his book was not a generalized hyperplasia but was localized in polypi of the fundus. Frankl (7) simply states that in cancer of the body, he did not find hyperplastic changes more frequently than usual. Novak and Martzloff (11), in their extensive study of hyperplasia endometrii, state: "In regard to cancer of the corpus or fundus uteri, we have observed in our laboratory only 1 case of cancer associated with endometrial hyperplasia, an almost negligible incidence. In this case, furthermore, the carcinoma, which was of ovarian origin, pushed into the endometrium from the outside. The endometrium played a purely passive rôle, so that this case has no significance as indicating a predisposition to cancer in cases of hyperplasia. We are convinced that no such predisposition exists."

A review of 22 adenomatous carcinomata of the body of the uterus seen at the Stanford University Hospital during the past 10 years reveals no case associated with hyperplasia of the endometrium. The following case, however, which was recently attended by one of us (S.), shows a coincidence of the two conditions in a very remarkable manner.

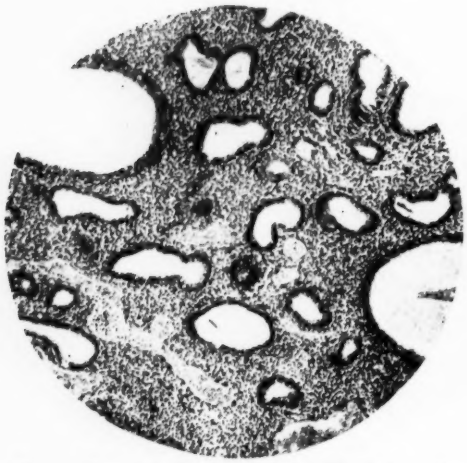

Fig. 1. Generalized hyperplasia of the endometrium showing numerous glands. (× 100.)

Fig. 2. Carcinomatous areas in hyperplastic endometrium. (× 100.)

Mrs. H. C. B., 44 years of age, ii-gravida, ii-para. First reported on March 1, 1928, complaining of uterine bleeding at intervals since January, 1928. The December period began on the twentieth, was normal in amount, and lasted for 3 days. The patient's youngest child was 12 years of age. The pregnancies had been normal with good recovery. No operations had been performed. Patient had lost no weight; appetite was satisfactory; bowel movements normal; bladder normal; and no leucorrhœal discharge.

Examination showed the perineum to be fairly firm; the cervix was soft and larger than normal; the uterus was in good position, movable, 1½ times normal size, firm, and smooth; the tubes and ovaries were normal. Speculum examination showed some odorless, bloody discharge; a large cervix, with no ulceration or erosion. It was decided to watch this patient until after her next menstrual period.

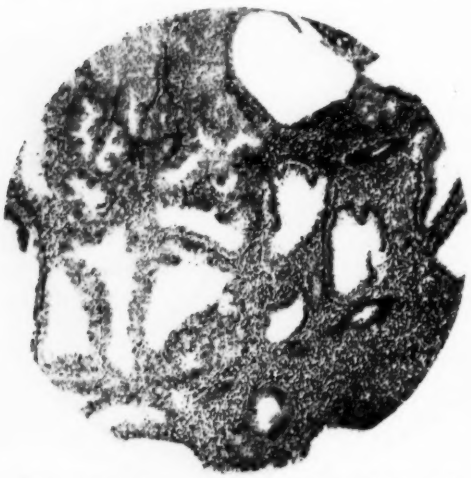

Fig. 3. Carcinomatous areas in hyperplastic endometrium. (× 100.)

The period began on March 11, lasted 1 week, stopped for a few days, then there was slight discharge again for 3 or 4 days. On April 16, there was a very profuse flow for 2 days with continuation of slight flow for 1 more week. May 8 the pelvic findings were unchanged.

On May 10 a dilatation and curettage was done under nitrous oxide gas anæsthesia. The uterus measured 10 centimeters. The canal was fairly well dilated; a No. 9 Hegar dilator entered readily. Many curettings were easily obtained. There was practically no bleeding and the patient returned to the ward in good condition.

The histopathological examination of the curettings showed glandular hyperplasia with one or two areas of definite adenocarcinoma. On account of this finding, a complete hysterectomy with removal of the tubes, ovaries, and cuff of the vagina was performed on May 12. No adhesions and no enlarged glands were found. Patient returned to bed in good condition and made an uneventful recovery.

Histopathological examination. Microscopic examination of the curettings reveals a picture of generalized hyperplasia of the endometrium (Fig. 1) with numerous glands, many dilated and cystic, and the stroma dense and cellular. There are a few dilated blood vessels, in some places an infiltration of free blood in the stromal tissue, but no marked inflammatory exudate.

Two fragments are seen in the curettings which are of especial interest. The first, measuring 5 by 2 millimeters, is made up almost entirely of malignant adenomatous tissue. The glands are of an immature type and there has been such a marked proliferation of the cancer cells that they present solid masses extending over several low-power fields. There is practically no stroma between these glands. At one extremity of the fragment is a small area showing hyperplastic stroma with several simple hyperplastic glands.

The second fragment measures 7 by 3 millimeters and is made up of hyperplastic endometrium in which are found various types of glands which immediately attract atten-

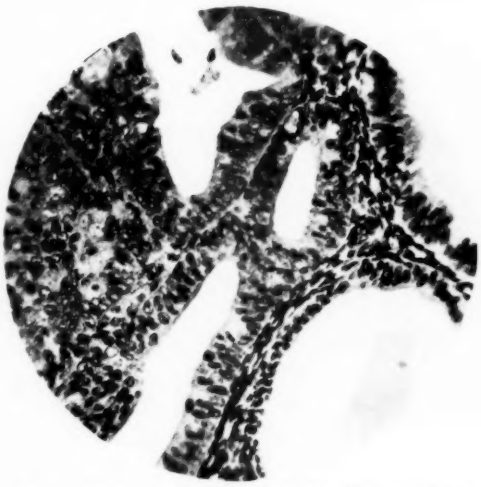

Fig. 4. Photomicrograph showing the close association of a solid nest of cancer cells, several carcinomatous glands, a normal endometrial gland, and a cystic hyperplastic gland. (× 500.)

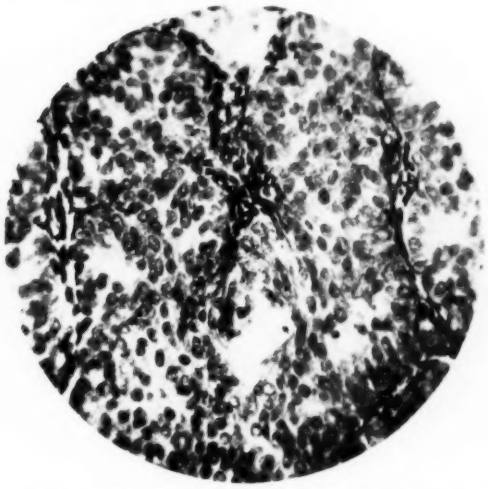

Fig. 5. Solid carcinomatous area in glandular cancer of the endometrium. (× 500.)

tion (Figs. 2, 3, 4). There are a few normal endometrial glands with high cylindrical epithelium such as are usually noted during the interval phase. Several cystic glands lined with a low cuboidal type of cell and with the lumen clearly outlined, are present in considerable numbers. There are also a few convoluted glands which are generally lined with a single layer of cells but are stratified in some places and in others send out little tufts or papillary projections into the lumen. The basement membrane of these glands is intact and their occurrence has been noted frequently in hyperplasia endometrii. The next type of gland seen apparently represents an early carcinomatous change in that there are occasional mitotic figures and stratification into from two to four layers, but the basement membrane is still intact. Finally, one finds definite carcinomatous glands with tremendous proliferation of the cellular elements, extension into the stroma, and the formation of dense masses of cancerous tissue in which the outlines of the individual cells cannot be made out (Fig. 5.).

Although all of the tissue obtained from the curettage was mounted and numerous sections were made, the two areas just described are the only ones showing unquestionable malignant changes. The only possible exception is a single gland found in a markedly hyperplastic area which presents staining irregularities and such stratification of the cells that it must be considered as at least "suspicious," if not definitely carcinomatous.

Gross specimen. The uterus is uniformly enlarged, weighs 180 grams, and measures 10.5 by 5.5 by 8.0 centimeters. It is of a soft consistency and does not present any other gross abnormality except a few small cystic structures in the myometrium immediately subjacent to the endometrium. These vary from the size of a pinhead to 4 millimeters in diameter and contain clear mucoid material. The endometrium measures from 2 to 4 millimeters in thickness, is irregular and shaggy, of a purplish color, and at the left cornu there is a small polyp 8 by 3 millimeters. The cervix is hypertrophied and contains a few small cysts. The fallopian tubes are normal. The left ovary appears normal except for a few small cysts containing clear yellowish fluid. The right ovary contains a blood filled cyst 4 by 3 by 2 centimeters in its medial pole.

Histopathology. Sections were taken from every part of the uterine cavity. On microscopic examination the first noteworthy feature is that no evidence of the malignant process seen in the curettings is demonstrable at any point. The appearance of the endometrium is consistent throughout the whole uterus. Owing to the previous curettage the superficial strata are absent and one sees merely a thin basal layer containing a few normal and many dilated and cystic glands (Fig. 6). The surface is represented by a thin layer of fibrin, blood clot, and inflammatory cells. The polyp noted grossly shows hyperplastic endometrium and may be an artefact resulting from the curettage. A few lymph "follicles" are noted in the endometrium and there is a slight round cell infiltration in some areas, but this is not a prominent feature. The myometrium shows some hypertrophy, but there are no myomatous formations. There is an extensive downgrowth of endometrial glands, some of which are accompanied by stroma, into the myometrium (Fig. 7). They are situated within 1 centimeter of the uterine cavity and since there is no circumscribed tumor the condition is regarded as an adenomyosis (Frankl) rather than an adenomyoma. Another striking finding is the presence of numerous large blood vessels, some of which show hyaline changes, both in the basal endometrium and in the myometrium immediately subjacent. The cervix presents chronic inflammatory changes with several cystic glands. The fallopian tubes show no abnormality. Both ovaries were studied in detail. A corpus luteum of recent origin could not be demonstrated in either ovary. However, each contains a number of small follicle cysts and there are numerous small hæmorrhagic areas in the stroma. The large blood filled cyst noted grossly in the right ovary was found to be lined with granulosa cells, many of which have undergone a transformation into lutein cells.

Histopathological diagnosis. (1) Hyperplasia of the endometrium. (2) Adenomatous carcinoma of the corpus uteri.

Fig. 6. Basal layer of hyperplastic endometrium 48 hours following curettage. (× 100.)

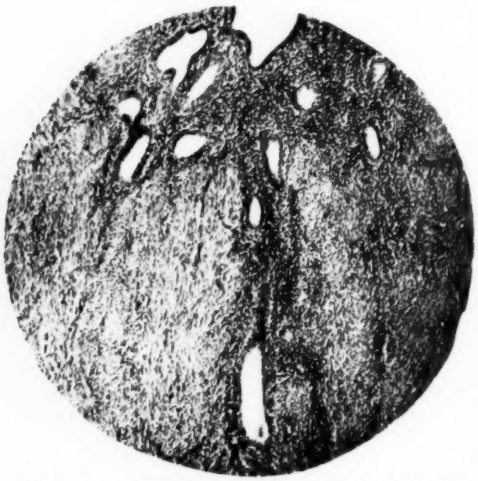

Fig. 7. Extension of endometrial tissue into the myometrium. (× 100.)

(3) Adenomyosis uteri. (4) Chronic cervicitis. (5) Follicle cysts of the ovaries.

We have thus demonstrated in this case a condition of marked hyperplasia of the endometrium accompanied by an adenomyosis uteri. In this endometrium was found an early adenomatous carcinoma which apparently was arising from previously simple hyperplastic glands. The only detailed description of such a process that we have been able to find is the case mentioned by R. Meyer. Another interesting feature presented by this patient is that apparently all the cancerous tissue was removed by the curette, a possibility which has already been reported by a number of observers.

Although this case is of considerable scientific interest, we feel that it can add little to our conception of hyperplasia of the endometrium. The condition is extremely common and its occurrence with malignancy of the body of the uterus is comparatively rare. As Novak and Martzloff assert, hyperplasia of the endometrium cannot be regarded as predisposing to a cancerous growth. However, the possibility of coincidence, no matter how slight, does exist and one must always bear in mind the importance of careful study of all tissue obtained from the uteri of women with abnormal bleeding at the time of the menopause.

SUMMARY

A study of the literature shows that only a few cases of hyperplasia of the endometrium associated with malignancy of the body of the uterus have been reported. This coincidence is thus regarded as very unusual. A case is described in which abnormal bleeding occurred in a patient at the menopause. The histopathological examination of the uterus showed an early adenomatous cancer arising in the superficial layers of a hyperplastic endometrium. All the malignant tissue was apparently removed by the curette. The hyperplasia was accompanied by an adenomyosis uteri.

Our thanks are due to Mr. Pierre Lassègues for the photomicrographs and technical work concerned with this study.

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FATAL EMBOLUS DUE TO INFLATION OF BLADDER WITH AIR[1]

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From the Department of Urology, St. Mary's Hospital

AIR embolus has long been recognized as the cause of alarming symptoms or death following the inflation of the bladder or urethra with air for diagnostic and operative procedures, yet its danger has not been sufficiently emphasized. It is still the common practice of many urologists of repute in the leading hospitals in the United States and abroad to employ air in inflating the urethra and bladder. Having recently experienced a fatal embolus following inflation of the bladder with air, I have attempted to ascertain its incidence by sending questionnaires to the members of the American, French, and Roumanian Urological Associations, to the well-known genito-urinary surgeons of Europe, and to members of the American College of Surgeons doing urological surgery. The results from this inquiry and from a perusal of the literature shows that its occurrence is rather common. For the purpose of preventing its occurrence in the future I present my case in detail.

Hospital No. 48454, male, age 56 years, president of a foundry, referred by Dr. E. Boldemann, entered St. Mary's Hospital, October 13, 1927, complaining of hæmaturia, inability to urinate, frequent urination, dysuria, and nycturia.

Patient's mother died of cardiovascular and renal disease; father, of apoplexy; and one sister, of scarlet fever. One brother is living but suffers from numerous varicosities.

Patient has had gonorrhœa but denies having had syphilis. He was circumcised when he was 25 years old and had neuritis when 51.

In January, 1927, the patient developed increased frequency of urination with nycturia. There was a sensation of fullness in the perineum, and at times the patient noted that the urine was turbid and had a foul odor. At this time, the size, force, and projection of the urinary stream became diminished, and he experienced the sensation of not completely emptying the bladder. This condition became progressively worse and required considerable straining to force out a few cubic centimeters of urine, until on October 11, 1927, complete retention took place. Two weeks later he developed a severe attack of hæmaturia passing bright red blood and a number of clots which cleared up in 3 days without any treatment. Three days later he developed a second attack of hæmaturia which convinced him that he should seek medical aid. No particular pain was experienced in the lumbar or sacral regions. In the past month he had lost 5 pounds in weight.

Physical examination. The patient was of the short, stocky type, and he had a thick neck. Auscultation of the heart revealed slight roughening of the first sound in the region of the tricuspid valve. The pulse was normal. In the region of Grocco's triangle the lungs presented an inspiratory wheeze terminated by small coarse râles. The blood pressure was systolic 166, diastolic 80. Examination of the blood revealed hæmoglobin, 90 per cent; erythrocytes, 5,030,000; leucocytes, 16,900; polymorphonuclear leucocytes, 85 per cent; small mononuclear lymphocytes, 11 per cent; large mononuclear lymphocytes, 4 per cent; coagulation time, 4 minutes. Wassermann reaction was negative.

In examining the blood 49 milligrams of urea per 100 cubic centimeters were found, 33 milligrams of non-protein nitrogen per 100 cubic centimeters, and 1.8 milligrams of creatinin per 100 cubic centimeters. The intramuscular phenolsulphonephthalein test yielded 70 per cent in 2 hours.

Urological examination. Tenderness and dullness were elicited in the suprapubic region, and although neither kidney was palpable considerable tenderness was found in the costo-vertebral angle on both sides. Rectal examination showed the prostate to be enlarged about five times its normal size; symmetrical, soft, and bound down laterally by adhesions. Cystoscopic examination revealed a bladder containing 250 cubic centimeters residual urine with a capacity of 300 cubic centimeters, intravesical encroachment of the middle and lateral lobes of the prostate, an ulcerated area on the left anterior octant of the bladder neck, and a round papillomatous mass about 4 centimeters in diameter in the base of the bladder. Plain roentgenograms were negative except for a small shadow in the left side of the pelvis presenting the appearance of a phlebolith. On account of obstruction of the outflow of urine caused by the enlarged prostate it was decided to remove the gland by the suprapubic route and, if feasible, resect the papillomatous mass at the same time. The usual suprapubic incision was made and the bladder inflated with 300 cubic centimeters of air with a syringe. A hissing sound (*sifflement*) was heard in the bladder during a period of a few seconds, and, as the silk guy sutures were inserted into the fundus of the bladder, the patient became cyanotic, the eyes fixed, the pupils dilated, and the pulse and respiration suddenly ceased. Immediately after, the heart beat could not be elicited. Caffein and camphorated oil were injected hypodermically; adrenalin directly into the heart; artificial respiration resorted to and oxygen introduced into the lungs. At the end of 30 minutes it was found that these efforts had been of no avail.

Necropsy. The patient was a well developed and nourished man. The pupils were equally dilated and the mucous membrane pale. The teeth were well preserved and presented numerous gold fillings. When the abdominal cavity was opened, small bubbles of air were found in the iliac, the mesenteric vessels, the vena cava, and in the renal veins. The lungs, liver, and the right chambers of the heart contained a coarse froth of air. The coronary arteries presented advanced atheromatous changes. The bladder showed considerable thickening of its wall, an intravesical encroachment of the prostate, ulceration of the neck, and a tumor mass in its base measuring 4 centimeters in diameter which had invaded the muscular coat and had presented necrosis in the center. Both kidneys presented a moderate degree of hydronephrosis.

The tumor mass located in the base of the bladder was removed. It was of a hard consistency and had invaded all three coats of the bladder wall. An irregular ragged greyish-white ulcer was found in its center under which there was a hard base. Microscopical sections of this tumor showed that the continuity of the epithelium was broken by necrosis

[1] Read before the Urological Section of the California Medical Association, May 2, 1928.

which had taken place in the center of the tumor. The surface epithelium was infiltrated by the tumor, which consisted of atypical glandular cells presenting many mitotic figures and a fibrous tissue matrix. In the latter a number of groups of atypical epithelial cells were seen. The glandular cells had formed numerous definite acini. A number of the blood vessels were found to have been invaded by the tumor cells. Diagnosis: adenocarcinoma of the bladder.

OCCURRENCE

Alarming symptoms and death due to the introduction of varying amounts of air into the circulatory system were early recognized by Morgagni, Claude Bernard, Bichat, and many of the earlier investigators. On the battle fields of the Napoleonic wars, soldiers were often observed to die from air embolism resulting from saber wounds of the neck. In 1818 Beauchesne reported the first authentic case proved by autopsy in which air had been aspirated in the course of an operation on a tumor of the neck. In 1883 Treves collected as many as 67 such cases adding two of his own in which air embolism was quickly recognized and successfully treated. Manauray reported a case occurring during an operation for fracture of the clavicle, and Depage and Courvoisier reported others accompanying the removal of new-growths of the neck in all of which the characteristic *sifflement* was heard. Davidson reported embolus following distention of the uterus with air, and Gordon, in discussing Senn's classical lecture on the subject, reported fatal embolism following the injection of air under pressure into an abscessed cavity of the pelvis, the negative pressure in the aspirating chamber having been accidentally substituted for positive pressure. Wolf is of the opinion that spontaneous emboli may result from a collection of gas beyond atmospheric pressure in an ulcerated stomach or diseased uterus. Revenstorf saw a fatal case result from suicidal cutting of the throat. Saugman, von Adelung, and Schlaepfer reported that it is not uncommon to find air embolism accompanying various diagnostic and therapeutic procedures of the chest. W. M. Spitzer encountered a fatal case which was due to the injection of the perinephric tissues with oxygen for diagnostic purposes. In 1903 Sick reported fatal embolism proved by autopsy following an attempt to dilate the bladder with a syringe in the course of operating on a carcinoma of the bladder. In 1913 Nicolich added another and Marion two cases, also proved by autopsy, in which embolus occurred following dilatation of the bladder with air in the course of performing a prostatectomy. Fox, Mark, Joly, and Ward reported cases of embolism after air inflation of the urethra in performing urethroscopy.

We sent 2050 questionnaires to the various surgeons of this country and abroad doing genito-urinary surgery, of which 791 were answered. A general preference was expressed for the use of water in inflating the bladder and urethra. A number of surgeons prefer water for distention of the bladder and urethra and use air only in making contrast cystograms. A number employed air alone, whereas a few surgeons used either oxygen or no inflation. The main objection to air seemed to be the likelihood of experiencing embolism from its use. Those who continued to use air preferred it for the reason that it is cleaner than water, because it does not run over and infect the operative field and perivesical tissues. A small number expressed their preference for oxygen, but its use seems to be as dangerous as air. A smaller number of emboli were reported following the distention of the bladder with aqueous solutions. All urologists who had noted untoward symptoms resulting from the employment of air were very emphatic in condemning its use.

ETIOLOGY

The presence of air under pressure in the normal bladder and urethra causes no harm as evidenced by the enormous number of cases in which it is being daily injected without the least signs of ill effects. The formation of emboli takes place by the entrance of air into the venous circulation either through an ulceration of the mucosa caused by some pre-existing pathological lesion such as an ulcer, a tumor, a deeply congested area due to cystitis, etc., or through a laceration of the mucosa caused by overdistention of the bladder. The veins that are particularly susceptible to the entrance of air are those whose walls are thickened or bound up in inflammatory material or those of a new-growth. If air is injected into a healthy bladder through a catheter, it will regurgitate back between the catheter and the urethral wall long before the mucosa becomes ruptured. On the other hand, if prostatic enlargement or stricture formation has caused tight approximation of the catheter to the urethra, increase in pressure can cause rupture of the bladder wall, the mucosa being the first to be lacerated. Once the veins of the bladder wall are ruptured, a minimum amount of pressure can cause penetration of air into the vesical veins and thence into the vena cava and right heart.

In discussing his case of fatal air embolus resulting from inflation of the bladder with air, Nicolich referred to the theory of Lewis, who thought that air entered into the circulation by way of the pelvis after having regurgitated up

through the ureters. Following this report Santini injected air under considerable pressure into the bladder of dogs and found that the healthy bladder invariably ruptured before air would enter the pelvis by way of the ureter. Air was then injected directly into the abdominal portion of the ureter and he reported that in this way it was possible to introduce air directly into the general circulation by way of the renal parenchyma. Shortly after, however, Poddighe was unable to confirm these observations. He injected air into the lumen of the ureter of 11 dogs under considerable pressure over a period of 15 minutes, 30 minutes, and longer, and found that although he was able to produce considerabe augmentation in the volume of the kidney, he was never able to produce death by air embolus. Careful autopsy of these dogs revealed huge dilatation of the pelvis and the tubular system of the renal parenchyma, with enormous compression of the glomeruli. The dilatation of the pelvis and tubules were responsible for the markedly increased size of the kidneys, but in no cases had the air entered the cardiovascular circulation. In dogs in which the veins of the bladder walls were traumatized, inflation of the bladder under minimum pressure caused death in a few moments, and autopsy revealed air emboli and frothy blood in the right heart.

Graves and Davidoff have shown experimentally that fluids may regurgitate from the bladder into the kidney by way of the ureters. The earlier investigations of Poirier and of Lewis and Goldschmidt, and the more recent work of Hinman and Lee-Brown on pyelovenous back-flow indicate that solutions are readily absorbed by the veins of the pelvis. In reporting recent research on the physiology of the ureter, F. Fuchs demonstrated that air can enter into the venous circulation of the calyces. This entrance of air is more likely if there is an ulceration of the mucosa of the pelvis due to some pre-existing pathological lesion or to ulceration due to overdistention. Such was not true in my case.

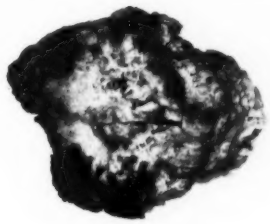

Fig. 1. Carcinoma of the bladder presenting ulceration of the overlying mucosa through which air entered the venous system causing fatal embolus. Actual size.

PATHOLOGY

Since the classical case of Beauchesne in 1818, the danger of air embolus has been emphasized in the teaching of surgery. Even before that time, Bichat and others believed that the entrance of a very small amount, even the smallest bubble of air, would be followed by very serious consequences. In 1885 Senn and in 1889 Hare reported extensive experimental studies showing that fairly large amounts of air could, in some cases, enter the veins without disastrous results. This was followed by the work of Goodridge, Larned, and others, who concluded that when an appreciable quantity of air entered the veins the result might be rapidly fatal, and by those of Blair and McGuigan who clearly demonstrated that this is particularly true when the air enters under pressure.

When air enters the right ventricle in even as small amount as 4 cubic centimeters, the arterial tension is lowered, the venous tension raised, and the contraction of the heart and the action of the lungs are considerably disturbed and are for a time rendered less efficient. These cardiorespiratory changes are uninfluenced by bilateral vagotomy (Quilliot). If a larger amount enters, this disturbance becomes increased giving rise to grave symptoms or death. Three theories have been advanced as to the cause of death—cerebral, pulmonary, and cardiac disturbances. Morgagni and Bichat advanced the first theory—embolus formation in the brain itself. Death in such cases was attributed to syncope resulting from anæmia of the vital centers of the bulb. Claude Bernard, Vulpian, Quilliot, Wolf, and others favored the second theory. They believed that air in passing from the right heart into the lungs formed veritable emboli which closed the various branches of the pulmonary artery resulting in death by suffocation. Magendie, Amussat, Depage, Goodridge, and others, in supporting the cardiac theory, attributed the grave symptoms arising from the introduction of air into the circulation to the lack of stimulus producing blood in the right heart, or to a direct deleterious reflex action of air in the heart itself. They explained that large amounts of air in the right heart reduced the intracardiac pressure to such an extent that it could not overcome the resistance of the pulmonary capillaries and that as the heart beat was of no avail, stagnation of the entire circulation soon resulted thus

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In discussing his case of fatal air embolus resulting from inflation of the bladder with air, Nicolich referred to the theory of Lewis, who thought that air entered into the circulation by way of the pelvis after having regurgitated up

through the ureters. Following this report Santini injected air under considerable pressure into the bladder of dogs and found that the healthy bladder invariably ruptured before air would enter the pelvis by way of the ureter. Air was then injected directly into the abdominal portion of the ureter and he reported that in this way it was possible to introduce air directly into the general circulation by way of the renal parenchyma. Shortly after, however, Poddighe was unable to confirm these observations. He injected air into the lumen of the ureter of 11 dogs under considerable pressure over a period of 15 minutes, 30 minutes, and longer, and found that although he was able to produce considerabe augmentation in the volume of the kidney, he was never able to produce death by air embolus. Careful autopsy of these dogs revealed huge dilatation of the pelvis and the tubular system of the renal parenchyma, with enormous compression of the glomeruli. The dilatation of the pelvis and tubules were responsible for the markedly increased size of the kidneys, but in no cases had the air entered the cardiovascular circulation. In dogs in which the veins of the bladder walls were traumatized, inflation of the bladder under minimum pressure caused death in a few moments, and autopsy revealed air emboli and frothy blood in the right heart.

Graves and Davidoff have shown experimentally that fluids may regurgitate from the bladder into the kidney by way of the ureters. The earlier investigations of Poirier and of Lewis and Goldschmidt, and the more recent work of Hinman and Lee-Brown on pyelovenous back-flow indicate that solutions are readily absorbed by the veins of the pelvis. In reporting recent research on the physiology of the ureter, F. Fuchs demonstrated that air can enter into the venous circulation of the calyces. This entrance of air is more likely if there is an ulceration of the mucosa of the pelvis due to some pre-existing pathological lesion or to laceration due to overdistention. Such was not true in my case.

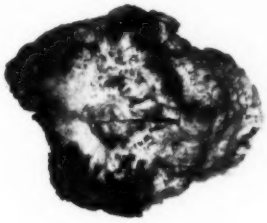

Fig. 1. Carcinoma of the bladder presenting ulceration of the overlying mucosa through which air entered the venous system causing fatal embolus. Actual size.

PATHOLOGY

Since the classical case of Beauchesne in 1818, the danger of air embolus has been emphasized in the teaching of surgery. Even before that time, Bichat and others believed that the entrance of a very small amount, even the smallest bubble of air, would be followed by very serious consequences. In 1885 Senn and in 1889 Hare reported extensive experimental studies showing that fairly large amounts of air could, in some cases, enter the veins without disastrous results. This was followed by the work of Goodridge, Larned, and others, who concluded that when an appreciable quantity of air entered the veins the result might be rapidly fatal, and by those of Blair and McGuigan who clearly demonstrated that this is particularly true when the air enters under pressure.

When air enters the right ventricle in even as small amount as 4 cubic centimeters, the arterial tension is lowered, the venous tension raised, and the contraction of the heart and the action of the lungs are considerably disturbed and are for a time rendered less efficient. These cardiorespiratory changes are uninfluenced by bilateral vagotomy (Quilliot). If a larger amount enters, this disturbance becomes increased giving rise to grave symptoms or death. Three theories have been advanced as to the cause of death—cerebral, pulmonary, and cardiac disturbances. Morgagni and Bichat advanced the first theory—embolus formation in the brain itself. Death in such cases was attributed to syncope resulting from anæmia of the vital centers of the bulb. Claude Bernard, Vulpian, Quilliot, Wolf, and others favored the second theory. They believed that air in passing from the right heart into the lungs formed veritable emboli which closed the various branches of the pulmonary artery resulting in death by suffocation. Magendie, Amussat, Depage, Goodridge, and others, in supporting the cardiac theory, attributed the grave symptoms arising from the introduction of air into the circulation to the lack of stimulus producing blood in the right heart, or to a direct deleterious reflex action of air in the heart itself. They explained that large amounts of air in the right heart reduced the intracardiac pressure to such an extent that it could not overcome the resistance of the pulmonary capillaries and that as the heart beat was of no avail, stagnation of the entire circulation soon resulted thus

TABLE I.—METHODS USED BY SURGEONS

	Number of Surgeons
Air distention (25,890 inflations)	34
Water distention (462,206 inflations)	464
Air and water distention	172
Oxygen distention	6

TABLE II.—METHODS USED BY UROLOGISTS

	Number of Urologists
Air distention	47
Water distention	588
Oxygen distention	6
No inflation	18

TABLE III.—EMBOLI AFTER INFLATION WITH AIR OR WATER

	Cases
After inflation of bladder or urethra with air	34
After inflation of bladder or urethra with water	14

causing death by lack of nutrition of the vital centers. Laborde and Frey concluded that death is brought about by a triple mechanism in which all three of the above theories play a rôle.

All investigators agree that small quantities of air can be introduced into the venous system at intervals causing slight or only transitory symptoms, due probably to the ability of the blood to absorb air, whereas if the total of these amounts were suddenly introduced under pressure grave symptoms, or death, might ensue. Marked difference in resistance to air embolism exist in different individuals and in different species. Hare observed that the introduction of 3 cubic centimeters of air into the venous system of three human beings caused no symptoms whatever, whereas Blair and McGuigan noted that the injection of 4 cubic centimeters of air into the circulation of dogs was followed by marked cardiac and respiratory changes. Delbet and Mocquat demonstrated that the coefficient of danger in dogs consists of the injection of 6 to 7 cubic centimeters of air per kilogram of body weight per minute. Rabbits and monkeys are particularly susceptible, while goats and some species of dogs are extraordinarily immune.

When air enters the right ventricle, it prevents the proper closing of the auriculoventricular valve on that side, and the light elastic air present in the vena cava readily allows regurgitation from the auricle. Each contraction of the heart causes the air to be churned backward and forward in the vena cava in the form of a coarse froth. The lack of the normal stimulus of blood in its cavities causes weakening of the contraction of the heart itself which is less efficient during this time. As a result of these two factors, little or no blood reaches the left ventricle and the whole circulation, including the coronaries and arteries supplying the vital centers in the bulb, suffer from lack of nutrition, resulting in respiratory failure, cardiac anæmia, and deterioration of the heart muscle. In every case, the respiration was found to cease first, the heart continuing to beat for some time after. If the circulation is re-established by artificial respiration, the vicious circle is broken; huge amounts of air may be disposed of by rapid absorption or elimination, after which the animal or patient is none the worse for his experience.

SYMPTOMATOLOGY

When air under pressure enters a vein of considerable size, a characteristic gurgling, hissing sound, or *sifflement*, may be heard. It is due to the entrance of air from the inflated bladder or urethra into the veins. When absorption takes place through a group of capillaries or by way of the renal pelvis, this diagnostic sound may be absent. No matter how small an amount of air has entered the heart by way of the venous circulation, there is a fall in the blood pressure, dyspnœa, and restlessness. This may be followed by syncope from which the patient soon recovers none the worse for his experience. Unquestionably these early symptoms are often overlooked or mistaken for other conditions. If a larger amount of air, even in some instances as little as 15 cubic centimeters has entered, the patient is seized with a sudden terror, coughs, becomes more dyspnœic, develops severe anæmia soon followed by cyanosis. The eyes become fixed, the pupils dilated. The patient may complain of nausea and an acute pain in the epigastrium and precardium. The respiration ceases, the heart action becomes irregular and often tumultuous, the pulse becomes more accelerated and rapidly sinks while the patient goes into profound syncope, which is terminated by convulsions of a tetanic character or a violent cough.

DIAGNOSIS

If, during the inflation of the urethra or bladder with air the patient suddenly complains of pain, becomes dyspnœic and cyanotic, develops a rapid pulse and heart action, accompanied by lowering of the blood pressure, and goes into syncope, air embolus should be suspected at once. The characteristic hissing sound, *sifflement*, may be absent, but when present is pathognomonic of the entrance of air into the venous system. Auscultation of the heart will often reveal the characteristic whir, *bruit de soufflement*, due to churning of air in the chambers of the right heart. Likewise, a number of mucous râles can

often be heard in the lungs due to the presence of air emboli. In some cases it is not uncommon to find local emphysema or infiltration with air of the tissues surrounding the urethra or bladder.

TREATMENT

If an air embolus is suspected, one should at once release the pressure under which it is being injected into the bladder or urethra. Many cases let alone will recover, but in order that any form of treatment shall be effective it must, in the majority of cases, be quickly applied. As it has been definitely proved by animal experimentation (Blair and McGuigan) that the heart continues to beat after respiration has ceased, artificial respiration in which pressure on the thorax is exerted during expiration should be resorted to and continued even while other measures may be used to resuscitate the patient.

On account of the rather deep position of the venous plexus draining the bladder, prostate, and urethra, direct withdrawal of blood from the veins containing bubbles of air, as advocated by Kleinschmidt, cannot be made. It might be stated in passing, however, that venesection is a valuable procedure particularly in those cases in which air has entered the veins of one of the dependent members of the body.

The usual cardiac stimulants consisting of the different forms of digitalis, caffein, camphorated oil, etc., should be employed. The best stimulant of all is the injection of adrenalin into the right heart itself. This consists of injecting 2 cubic centimeters of 1:1000 adrenalin solution through a fine needle that has been pushed through the chest wall and lungs at the anterior extremity of the right third or fourth intercostal space. Opening of the thorax and direct massage of the heart, although drastic, has been used with success.

In 1910, von Lesser attempted to sweep the air from the right heart into the pulmonary circulation so that the impact of fluid against the tricuspid valve cusps would cause them to close in the normal manner. He, therefore, employed simple infusion of 0.5 per cent sodium chloride solution and reported good results from its use. In experimenting on animals Blair and McGuigan, and others, not only found its administration of no benefit whatever, but actually dangerous, because the already weakened heart tends to dilate if additional fluid is added to the circulation.

The most rational form of therapy is the prevention of entry of air into the venous system by the abandonment of its use in inflating the urethra and bladder for diagnostic and operative purposes. One should abandon the common practice which consists of injecting analgesic or antiseptic solutions into the bladder by forcing open both sphincters with an air cushion obtained by compressing the bulb of the common asepto-urethral syringe. Water can be readily substituted for air for the purpose of inflating the urethra and bladder in performing cystoscopies and in making cystograms. Antiseptic solutions such as mercurochrome, rivanol, boric acid, etc., can also be used in place of air in inflating the bladder to facilitate its surgical attack.

DISCUSSION

In reviewing the factors that led to a fatal termination of the case reported herein and which might have happened in any patient in whom the bladder had been inflated with air, I wish to emphasize the following points: The ulcerated mucosa overlying the adenocarcinoma that was found to have been present in the base of the bladder offered an excellent portal of entry for air into the veins of this new-growth. Had the surface of the vesical mucosa been intact, the small amount of pressure utilized in inflation of the bladder would never have caused air to enter the venous circulation. Increase in intravesical pressure was favored by the encroachment of the enlarged prostate which prevented regurgitation of air to the outside between the wall of the prostatic urethra and the catheter. The probability of the entrance of air into the circulation by way of the pelvis, after having ascended the ureters from the bladder, was not likely to have occurred in my case because the characteristic *sifflement* produced by the entrance of air into the veins of the bladder was heard. The advanced sclerosis of the coronary vessels might have aided in stagnation of the cardiac circulation causing anæmia and deterioration of the cardiac muscle and favoring sudden arrest of the heart action because neither the heart beat nor the pulse was perceptible two seconds after the patient had become convulsed and cyanotic. It is also probable that the air might have passed through the lungs into the cerebral circulation thus causing anæmia of the respiratory center in the bulb producing respiratory failure. These factors, which coupled with the patient's familial and individual predisposition to the formation of gaseous emboli, which varies enormously in individuals and in different species, were responsible for the sudden fatal issue.

SUMMARY AND CONCLUSIONS

1. Distention of the bladder or urethra with air or oxygen for any purpose may result in

TABLE IV.—SUMMARY OF CASES

Reference author	Sex and age	Diagnosis	Diagnostic or operative procedure	Pathological lesion favoring air embolus	Symptomatology	Physical signs	Treatment	Results	Autopsy
P. André*	Male 57	Vesical calculus	Inflation bladder 600 c. cm. air for removal of calculus	Advanced cystitis with contraction of bladder		Sudden cyanosis and cessation of respiration	Artificial respiration	Sudden death	Note: Previous cystolithotomy successfully performed under water dilatation.
E. G. Ballenger*	Male 70	Prostatic hypertrophy	Cystography	Traumatized posterior urethra in passing catheter by assistant	Fear, dyspnœa	Collapse	Artificial respiration, oxygen stimulants	Death in a few minutes.	
L. Bazet*	Male 62	Adenoma of prostate	Inflation bladder with air, prostatectomy		Dyspnœa	Dyspnœa, rapid pulse, death		Death in 5 hours.	
W. Braasch* three cases	One female and two males	Bladder tumor	Cystography and cystoscopy with air inflation	Bladder tumor	Dyspnœa, general malaise	Crepitation of suprapubic tissues	Application of heat, general stimulants	One death and two recoveries.	
A. Chute*	Male 40		Urethroscopy, air inflation deep urethra	Unknown	Sensation of air moving in abdomen, faint feeling	Rapid and weak pulse, unconsciousness		Recovery.	
Wm. Fox	Male	Urethral stricture	Urethroscopy with air	False passage in urethra due to former instrumentation	Sensation of something running down thigh	Swelling in perineum and thigh		Recovery.	
A. Goldstein*	Male 58	Carcinoma of prostate, vesical calculi, gangrenous cystitis	Inflation bladder with 200 c.cm. air for prostatectomy	Grayish ulceration of bladder, carcinoma of prostate		Sudden death		Death.	
B. Guisy*	Male 40	Bladder tumor	Inflation bladder with air for removal of tumor	Bladder tumor	5 hours after operation; syncope, dyspnœa	Lowering of blood pressure, rapid pulse		Death in 1 hour.	
G. Marion	Male 62	Prostatic hypertrophy	Inflation bladder 150 c.cm. air, prostatectomy	Tear in vesical mucosa. Cancer of right kidney	Rapid pulse, syncope	Emphysema of perivesical tissues, cyanosis		Death	Air in right heart, renal veins and inferior vena cava. 500 c. cm. in all.
G. Marion	Male	Prostatic hypertrophy	Inflation bladder with air, prostatectomy	Not given	Rapid respiration, syncope			Death	No autopsy.
E. G. Mark	Male 53	Infected follicle of urethra	Urethroscopy with air	Incised urethral follicle	Pains in epigastrium cough, cyanosis, disappearance of pulse	Whirr over heart, dilatation of pupils, dilatation of sphincters	Artificial respiration	Recovery next day.	
A. Meads*	Male 20	Urethritis	Forcing of argyrol into posterior urethra by air cushion	Abrasion of urethral mucosa by passage of sound	Gurgling, pain in left chest, loss of vision, dyspnœa and collapse	Dilatation of pupils, rapid pulse	Artificial respiration, cardiac stimulants	Recovery in 20 minutes	

*Personal communication.

TABLE IV.—SUMMARY OF CASES

*Personal communication.

TABLE IV.—SUMMARY OF CASES—Continued

Reference author	Sex and age	Diagnosis	Diagnostic or operative procedure	Pathological lesion favoring air embolus	Symptomatology	Physical signs	Treatment	Results	Autopsy
J. L. Morgan*	Male 35	Diverticulum of bladder	Cystogram injection of neosilvol *per urethram* by air cushion	Obstruction in membranous urethra	Pallor, dyspnœa, perspiration	Rapid weakened pulse		Death in 9 hours.	
G. Nicolich	Male 73	Prostatic hypertrophy	Inflation bladder 300 c.cm. air, prostatectomy	Dilated veins in bladder and prostatic urethra	Precordial pain, nausea	Sudden arrest of respiration and pulse after cough		Death	Bubbles of air in right ventricle and vena cava.
F. Patch*	Male 72	Hypertrophy of prostate	Inflation bladder with air, prostatectomy			Sudden death		Death	
Sick	Male 60	Bladder carcinoma	Resection of carcinoma, air inflation	Bladder carcinoma	Sudden shrieking, dyspnœa	Cyanosis, weakened pulse, collapse	Artificial respiration	Death	Air in right heart and inferior vena cava
L. D. Simon*						Gurgling sound in bladder		Death	
A. R. Stevens*	Male 65	Papilloma of bladder	Cystography	Bladder tumor	Chill, precordial pain, dyspnœa	Rapid pulse, collapse		Death.	
J. N. Vander Veer*	Male 63	Carcinoma of bladder	Inflation bladder with air for cystotomy and fulguration	Sloughing, bladder carcinoma	Dyspnœa, collapse	Rapid pulse, death	Artificial respiration, oxygen and stimulants	Sudden death	
	Female 54	Carcinoma of bladder	Partial cystotomy	Sloughing, bladder carcinoma	Sudden death	Gasp, death	None	Death in 48 hours.	
V. G. Vecki* Witnessed at Budapest	Male 29	Urethral stricture	Urethroscopy with air		Dyspnœa	Rapid pulse, collapse		Revived after collapse, death 6 hours later	Nothing noted at autopsy.
A. Wallace*	Female 56	Bladder papilloma	Fulguration of papilloma in bladder partially filled with air	Papilloma of bladder	Explosion sound, gurgling	Cyanosis, syncope, dyspnœa, rapid pulse, pallor, lowering blood pressure	Oxygen, caffein	Recovery in 15 minutes	
H. Walther*	Male 85	Bladder carcinoma	Cystotomy, air inflation	Vascular carcinoma of bladder				Sudden death.	
R. Ward*	Male 68	Urethral stricture	Urethroscopy with air	Laceration of mucosa by tight fitting urethroscope	Cyanosis, dyspnœa, convulsions	Rapid pulse	Artificial respiration, diaphragm incised and massage of heart	Death	Air in right ventricle, aorta, circle of Willis, pulmonary vein, inferior vena cava. No air in bladder.

*Personal communication.
Additional cases were experienced by Wolbarst, Sir Wm. Kingston, Mr. Joly (3), and J. Abrams, but no exact data could be obtained.

transitory or grave symptoms should it enter the venous circulation.

2. A fatality is herewith reported which was proved at autopsy to be due to air embolism following the inflation of the bladder for an operative procedure.

3. Increased intravesical and intra-urethral pressure is favored by prostatic hypertrophy, as was noted in this case, or by stricture formation, which prevents the escape of air between the walls of the urethra and the indwelling catheter or cystoscope. Rupture of the vesical mucosa by overdistention, or the presence of a pre-existing pathological lesion such as marked inflammation, ulcer formation, or a new-growth weaken the bladder wall thus favoring the entrance of air into the venous circulation.

4. Undoubtedly mild symptoms consisting of restlessness, transient changes in the respiratory and cardiac action have been overlooked, as has also the cause of fatal termination in such cases. Death is due to the arrest of the pulmonary circulation, to gaseous distention of the right heart thus preventing function of the tricuspid and pulmonary valves, to little blood reaching the left ventricle, so that anæmia of the vital centers of the brain is produced, and to stasis of the coronary vessels.

5. The most effective treatment of air embolus is the immediate release of air pressure in the bladder, artificial respiration, and injection of 2 cubic centimeters of 1:1000 adrenalin solution directly into the right heart.

6. Air in the bladder and urethra should be used with the greatest caution. Inflation of the urethra and bladder with air for diagnostic, therapeutic, and operative procedures should be abandoned and harmless sterile water or mild antiseptic solutions substituted.

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EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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MARCH, 1929

CRANIAL INJURIES

WE have never had a very clear understanding of the pathology underlying concussion and contusion of the brain. There is one factor, however, that until recent years has not received sufficient attention. It seems reasonably certain that after a blow on the head sufficiently severe to produce unconsciousness, there is a primary anæmia lasting for a very brief time followed by œdema, which may be very slight and easily absorbed. If there should be more than an easily absorbable amount, an increase in intracranial tension is brought about, since the brain in adults is inclosed in a rigid skull.

This acute increase in intracranial tension then becomes of prime importance, meaning life or death to the patient. All head injuries should be managed so as to bear this essential feature in mind. Unfortunately, the picture is often complicated by a secondary factor which commonly receives more than its share of attention in routine hospital work. I refer to such complications as fracture of the skull itself, extra- or intra-dural hæmorrhage, infection, etc. Each of these complications presents definite, well known indications for treatment.

In 1924, my associate, Dr. B. B. Neubauer, and I felt that it would simplify the management of head injuries if we could classify such injuries as a whole, according to what we had come to believe was their essential feature—intracranial tension. Our cases readily divided themselves on this basis into three groups: (1) no increase in tension; (2) moderate increase in tension, and (3) marked increase in tension.

At that time, we were doing many spinal punctures, finding that the cerebrospinal pressure reading fitted the clinical picture so regularly in these three groups, that we now reserve spinal puncture for the cases concerning which there is any diagnostic doubt or in which it is desirable to use it as a therapeutic measure. The details of the varying clinical pictures as they progress from no increase to a marked increase in tension, with the management of each of these groups, may be found by those interested, in our original article on this subject.[1]

Since this time, others have taken similar viewpoints in the management of cranial injuries. We have found this plan, based as it is on the control of this all essential feature —intracranial tension—greatly simplifies the problem, which I must confess was often quite confusing, when we were thinking in the terms of concussion, contusion, and compression.

The majority of head injuries, about 70 per cent, will fall into the milder groups of increased tension and will respond to the

[1] Ann. Surg., 1924, lxxix, 481.

non-operative plan of management if no complicating factor arises. Of the remaining 30 per cent about one-half will also respond to non-operative treatment, but in our experience some 15 per cent will fail to do so. In the latter group, we feel sure that subtemporal decompression is indicated and in some cases it will unquestionably relieve tension sufficiently to tide the patient over the emergency. It is of prime importance that the patient be kept under observation and treatment for a sufficient length of time. We believe that such sequelæ as headache, dizziness and even Jacksonian epilepsy, may be averted if this be done.

J. Stewart Rodman.

SOME RECENT EXPLORATIONS IN THE FIELD OF VISCERAL NEUROLOGY

THE exceedingly complex nature and the remarkable autonomy of the physiological processes has fascinated man from the earliest times. How much accurate knowledge of the physiology of the nervous system has been lost with the passing of ancient peoples, as is maintained by some writers to be the case, we do not know. However, we do know that within the period of recorded history, our knowledge of this subject has grown by exceedingly slow degrees. No sooner has an apparently established fact been accepted than it has had to be abandoned. It is with reluctance that we ever admit the uncertainty of our position and retrace our steps to where we started. Fortunately, for our self-respect, however, we can usually find a new foothold as we relinquish the old. The very spirit of science forbids us to mark time knowingly.

It is not so long since it was first observed that stimulation of the vagus slackened the pace of the heart. Such a result was beyond the comprehension of our medical forbears. Notwithstanding anatomical facts to the contrary, many of them weakened and denied that the vagus nerve had any connection with the heart. Indeed, most of us fall into the ways of Hamilcar's ancient pilot who, when compelled to report the loss of his fleets to his stern master, offered the excuse that blood-red toads and seaweed filled the horizon.

We continue to pursue the study of physiology with rapt interest, and sometimes not without dismay. At the present time, a Magellan of physiology, who now has reached the ominous age of 78 years, holds the attention of Europe. Pawlow's new books *Conditioned Reflexes* and *Activity of the Cerebral Hemispheres*, translated by one of his former pupils, Anrep, of Cambridge, will soon appear in English.

Pawlow's earlier work on gastric, pancreatic, and salivary fistulæ, and the observations made on his "miniature stomach" are well known. The more recent investigations of his school have been sketched in outline by Gantt.[1]

Pawlow considers all acts as reflex, and distinguishes between inborn reflexes and acquired psychic reflexes. Noting the psychic flow of saliva, he sought a method of measuring this psychic activity. In order to eliminate every external stimulus, he had his laboratory surrounded by a moat several feet deep, filled with sawdust to intercept vibrations from the street. The working rooms were widely separated, built of walls 2 feet in thickness, and guarded by iron doors padded with rubber. Dog and operator were likewise separated. The dog's cell consisted of two shells of concrete, the inner one suspended by a huge iron hook within the outer one. By

[1] Gantt, W. H. Recent work of Pawlow and his pupils; conditioned reflexes; sympathetic nervous system (Orbeli); epilepsy and cerebrospinal fluid (Speransky). Arch. Neurol. & Psychiat., 1927, xvii, 514–528.

means of an electric switchboard, the experimenter communicated with his subject and could give the conditioning stimulus: skin irritation, light, odors, and sounds; he could also feed the dog without being seen, and could observe his subject through a periscope. The response of the dog was determined through the flow of saliva, which was measured by a manometer and recorded on a drum. Usually, food was used as the unconditioned stimulus but an electric stimulus to the paw could be used, in which case the corresponding foot was suddenly raised at the proper time after the conditioning stimulus had been applied.

The conditioned stimulus, for example light, must be given before the unconditioned stimulus, for example, food, and the two must be associated 20 or more times, depending on the animal and other factors, before the application of the conditioned stimulus alone suffices to produce the result, for example, a flow of saliva.

Some interesting psychic responses were noted. For example, if a circle was presented as a conditioned stimulus (followed by food) and an oval as a negative conditioned stimulus (not followed by food), and later a figure midway between the two was presented, the animal might whine, refuse to eat, become drowsy or excited, and present an array of symptoms referred to by the writers as neuræsthenic.

Other animals, such as fish, may be used, and it is interesting to note how well these dull pupils distinguish between red and green lights. Mice may readily be conditioned in such a manner that they will scamper into the "dining room" at the ringing of a bell, just as the pigeons of Venice come flying to the piazza of St. Mark's from all points of the heavens at the stroke of eleven, which for centuries has been the hour of feeding.

Krasnogorski, one of Pawlow's older pupils, Ivanov-Smolensky[1], and others have found, in their study of conditioned reflexes of children, that the skin analyzer begins to function at 3 months, and that infants can distinguish between the odors of camphor and cologne at 8 months. They have also observed that an idiot may have the level of a fish and that neurotic children may develop and lose conditioned reflexes more quickly than normal children. This work has been extended to other psychiatric conditions.

Whether this work and other researches carried on in Russia will cease with the death of the master, as has been freely voiced in several European clinics, remains to be seen.

Workers on the physiology of the nervous system elsewhere, however, are not idle,[2] and the tremendous impetus given to the study of the visceral nervous system by the work of Hunter and Royle has resulted in the opening of an entirely new vista in surgery, which promises to equal in importance and usefulness the crowning achievements of general surgery of today.

Raynaud's disease has defied satisfactory treatment for years; today the pain and gangrene, in their usual manifestations, may be relieved at once by sympathetic neurectomy.[3] Thrombo-angiitis obliterans held its victims and gave no quarter; now the torturing pain, at least, may be made to yield at once in cases selected according to the vasomotor response and the ulceration usually shows some improvement.[4] In Japan, where leprosy is rampant, it has been found, as Professor Shinosacki informed me, that acral pain and

[1] Ivanov-Smolensky, A. G. On the methods of examining the conditioned food reflexes in children and in mental disorders. Brain, 1927, l, 138–141.

[2] Harris, H. A. Vascular diseases and the sympathetic system. Brit. M. J., 1927, i, 789–792.

[3] Adson, A. W., and Brown, G. E. Treatment of Raynaud's disease by lumbar ramisection and ganglionectomy and perivascular sympathetic neurectomy of the common iliacs. J. Am. M. Ass., 1925, lxxxiv, 1908–1910.

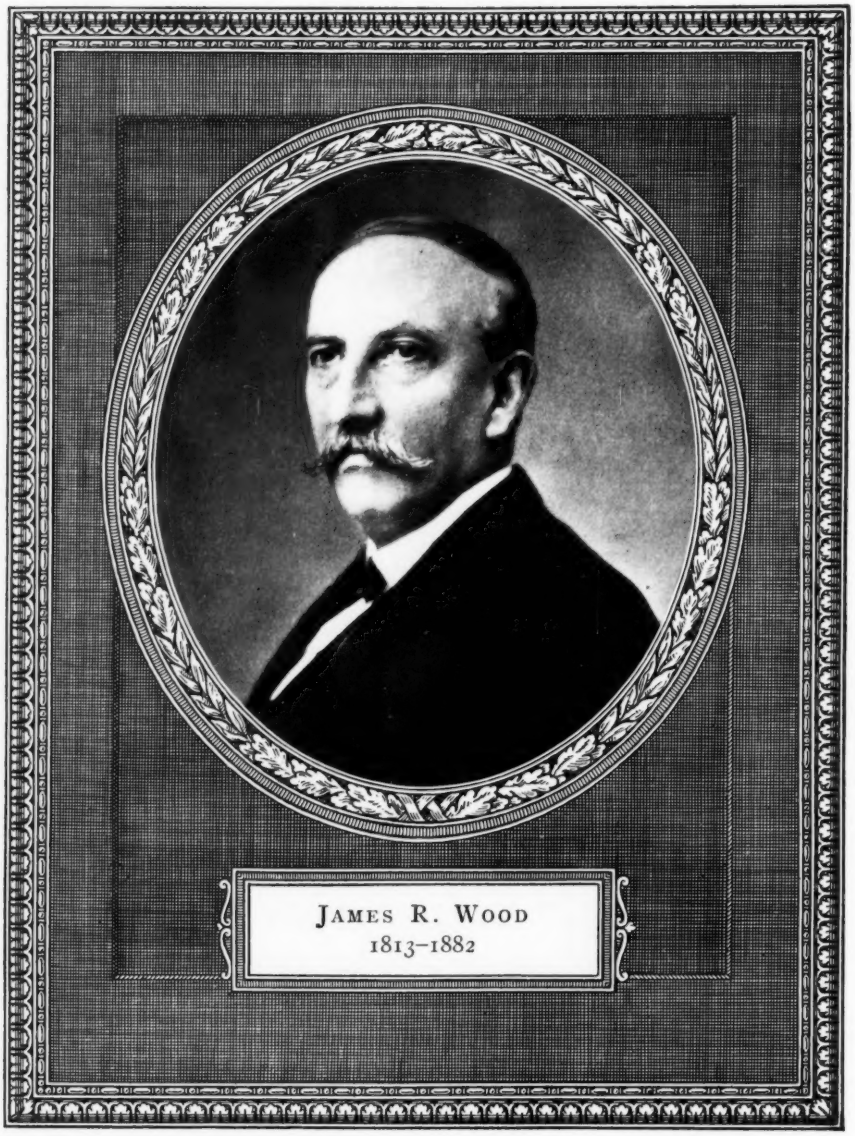
[4] Adson, A. W. Surgical relief of Raynaud's disease and other vascular disturbances by sympathetic ganglionectomy and perivascular neurectomy. Ann. Clin. M., 1926, v, 161–167.

gangrene, which are not uncommon, may be combated by sympathetic neurectomy and ramisection, thus affording these unfortunate patients much comfort. It is hardly an idle speculation that essential hypertension,[1] bone disturbances such as osteoporosis,[2] and other disorders which so far defy our best efforts will some day be relieved.

[1] Leriche. Relations between the sympathetic nerve and bone disorders. Foreign letters, Paris. J. Am. M. Ass., 1927, lxxxix, 633.
[2] Rowntree, L. G., and Adson, A. W. Bilateral lumbar sympathetic ganglionectomy and ramisectomy for polyarthritis of the lower extremities. J. Am. M. Ass., 1927, lxxxviii, 694–696.

The surgeon who would advance this frontier, however, must be thoroughly familiar with what is known and what is unknown of the anatomy, the physiology, and the pathology of these structures; he must possess wide technical knowledge and the ability and patience to use it; he must be courageous but not foolhardy, and must be ready to view his own disappointments and those of his colleagues with a spirit of co-operation and charity.

HENRY W. WOLTMAN.



JAMES R. WOOD
1813–1882

MASTER SURGEONS OF AMERICA

JAMES RUSHMORE WOOD

JAMES RUSHMORE WOOD was born at Mamaroneck, New York, on the 14th of September, 1813. Surgeons operating in this year of grace 1929 have little idea of the chaotic state of surgery not only in America but throughout the whole world in those pre-anæsthetic and pre-aseptic days. When Dr. Wood began his medical work in 1829, just one hundred years ago, surgery was beginning to be recognized as a science, for it was not until after 1800 that the surgeon began to have recognition or any standing whatever. It was in that year, after much difficulty, the Royal College of Surgeons of England obtained its first charter. In the House of Lords even at this time it was said in open discussion that "there is no more science in surgery than in butchering."

"It is only necessary to remember that a little over a hundred years ago there were scenes enacted in the name of surgery which eclipsed in horror the frightful cruelty of the Spanish inquisition, the untold miseries of the Bastille, the indescribable sufferings of the Black Hole of Calcutta, and the excruciating pains of the Turkish bastinado and the cruel massacre of the Huguenots. . . . Patients were held down upon the operating table by brute force and were operated upon while in the full possession of their senses; they were heard to cry out in heart-rending screams for a discontinuance of the tortures; they were incised with red hot knives, and they were compelled to have their wounds dipped in a caldron of seething tar to control hemorrhage." (Dennis.) This is quoted only to impress upon our minds that the call to be a surgeon in the year 1829, when Dr. Wood began his career, must come to a man of unusual qualities. The times were to us unbelievably backward, for at this date the large cities in the United States were even using tinder and flint to light their fires, and the first railroad did not operate by a steam locomotive until 1831.

But Dr. Wood was a man of unusual qualities. We cannot judge him by our present-day standards. We must judge him by the use of the tools and by the environment that obtained in that age and after we have studied his work, his accomplishments and his great fame—for he was *the* famous surgeon of *the* famous (Bellevue) hospital in America in his day and generation. Surely we can, after reading of his life, say truly,—Here was a *man*, and—Here was a master surgeon!

Dr. Wood's family were Quakers, a sect that has often produced genuine men. He had meager schooling. He never enjoyed a college education. He attended his first course of medical lectures guided by his preceptor, Dr. David L. Rogers, at the College of Physicians and Surgeons, located in Barclay Street, New York, about the year 1830. Can any of us who know the present College of Physicians and Surgeons as the great "Medical Center" now sweeping the skies at 168th Street, New York, imagine a medical college in Barclay Street! But he graduated at Castleton, Vermont, in 1834. Dr. Wood then began his practice in the Bowery, but fire soon involved the destruction of his home and all his books, instruments, and specimens. This was a serious loss to him. But as Osler wrote to Trudeau after a similar experience and disaster: "Dear Trudeau: I am sorry to hear of your misfortune, but take my word for it there is nothing like a fire to make a man do the Phoenix trick." And Dr. Wood did it. He moved his office to Broadway and he married Miss Emma Rowe, daughter of James Rowe, a retired merchant, and in due time had one son and two daughters. His practice grew apace. Aside from his general practice and his surgical work, he brought into the world many who afterward became our leading financiers and citizens.

Dr. Valentine Mott, Dr. Willard Parker, Dr. Alonzo Clark were prominent contemporaries.

Dr. Wood will always be associated with Bellevue Hospital. This celebrated institution goes back even to the date of 1658, and is noteworthy as being *the first hospital in the civilized era of American history*, when the city of New York numbered only one thousand souls! The first fifty years of the nineteenth century are not happy years to record for this institution. "The four wild horsemen of Death, Yellow Fever, Smallpox, and Typhus Fever dashed back and forth over the young institution. . . . It was difficult to provide adequate nursing. As convicts and insane were kept in the same institution, the insane patients were nursed by the convicts" [a wonderful arrangement!]. At one time out of 54 confinements, 28 died! What do we think of such mortality as this: phthisis, 74 per cent; delirium tremens, 25 per cent; puerperal fever, 85 per cent?

At this time there arose a young man, aged 34, Dr. James R. Wood, who, with the co-operation of Dr. Willard Parker and Dr. Metcalf, saved the doomed institution, for an indignant city was about to pull it down. By his courage and industry he swept the Augean stable. He had ever been a keen politician (an intimate friend of Henry Clay), and he knew how to manage the politicians, for Bellevue was then a political "job."

One of the great services which Dr. Wood rendered to medicine was that he was chiefly instrumental in the passage of the act granting for anatomical teaching the bodies of all vagrants dying unclaimed. His position as demonstrator of anatomy in 1837 enabled him to see the wisdom for this act, as previously medical

students had been compelled to be known as "body snatchers," or as our Scotch confrères termed it, "resurrectionists."

Dr. Wood's celerity in operating was acquired in the days before ether or any anæsthetic (except alcohol) was employed. He practiced surgery for 10 years before 1844. He quickly learned to cut with equal skill and precision with either hand. He took more than a little pride in his speedy work. Frederick S. Dennis, who was later associated with him as a partner, and is still living in New York, in good health, says that Dr. Wood could amputate the thigh in *nine seconds!*

One of Dr. Wood's house surgeons in the early days at Bellevue, also still living in New York, Dr. Henry Mann Silver, writes that "James Rushmore Wood was a man of wonderful personality; a great anatomist; a rapid and skillful operator whose results were brilliantly successful. He had great powers of clinical observation and diagnostic acumen. His energy was unfailing and he was always on the alert to detect and combat any unfavorable sign. The tripod on which he rested his treatment was rest, cleanliness, and free drainage. Although stern and unyielding on the professional side, he always carried with him the warm and helpful side for all those worthy of it. His house staff, private students, and patients adored him. He was an inspiration never to be forgotten." A wonderful tribute!

Now before we review his particular and special contributions to surgery, let us read what he did for humanity and the nursing profession while he was at Bellevue Hospital.

In 1869 was inaugurated at Bellevue Hospital the *first ambulance service for cities*. Although Dr. Dalton was the chief mover in this service, he could never have accomplished it without the backing of James R. Wood, then practically chief of staff. Dr. Wood brought to bear his wonderful personality and his "pull." "This ambulance service was so perfected in discipline and detail that it has been but little changed to this day. The system has been adopted by the hospitals of the world." (1869.)

A few years later, in 1873, greatly by the efforts of James R. Wood, another record maker was accomplished. The first training school for nurses was inaugurated for all America. Few will deny that this was an epochal event. Helping to start the undying life of the mother of all training schools should give undying luster to the fame of any man. Bellevue opened its Training School May 1; New Haven, October 1; Massachusetts General, October 1 (1873). How many training schools for nurses are there to-day? And how many surgeons must almost abandon their operative work without the help of the trained nurse!

Dr. Wood had an individual personality. Like some other great men we know, he was not averse to the spectacular. His students ever called him "Jimmy Wood." It was not a term of disrespect, but only one of affection.

"Before he entered the operating room he used to put on his long black gown over his street clothes. This gown was black so as not to show former splashes of blood and was buttoned tightly about his neck and wrists. On this gown above his heart Dr. Wood always pinned a red rose or carnation. Cheers always welcomed his dramatic appearance." His clinics began attended only by the orderly and one student. Later it was not unusual to see over a thousand students and doctors in attendance. He frequently almost emptied the clinics of other colleges and hospitals in New York the day he operated, so popular and instructive were his clinical lectures and his surgery. It is with no disrespect to tell, furthermore, that his operating gown would often be festooned with needles threaded with waxed silk (usually kept nobody cared where, as long as they were at hand when required). As before stated, Dr. Wood's early education had been meager, but he felt that the dignity due to the profession required an occasional Latin phrase. One who heard him say it has told the writer that he would at times, in admonishing his students to do meritorious work, say to them most sententiously: "Remember that the eyes of the *vox populi* are always upon you." Few of us are without faults, but few are loved for them as was James R. Wood.

"Dr. Wood from the beginning of his connection with Bellevue in 1847 began to collect postmortem material with the intention of founding a museum. As an aid in the accomplishment of this object he offered prizes for the best anatomical dissections . . . he presented this collection in 1856 to the New York Commissioners of Public Charities and Correction." Thus was founded the *Wood Museum*. Dr. Willard Parker remarked that "the Wood Museum as it now stands is the grandest monument ever erected to any surgeon in this country"; and the London *Lancet*, speaking of its rich collection of antique specimens, said: "It is not a little remarkable that this museum like our own Hunterian owes its origin to a distinguished surgeon whose work is known all over the world, including especially some of the most beautiful and successful instances of operation for the reproduction of bone."

"In periosteal reproduction of bone, Dr. Wood had an international reputation . . . and the renowned Langenbeck in an address said that he did not believe a corresponding preparation really existed anywhere (after a specimen of a regenerated lower jaw had been shown by Dr. Wood in 1877 before the German Congress of Surgeons in Berlin)." England gave him recognition when the London *Lancet* at about this time said, editorially: "Dr. Wood is entitled to great praise for having been one of the pioneers of periosteal surgery." American surgeons know only too well that neither English nor German surgeons were in the habit in those days of throwing bouquets to American doctors.

Dr. Wood excelled in cutting for stone in the bladder. Surgeons would flock to New York just to see him do this operation. He invented an instrument—a

"bisector"—which he used with precision and dispatch. It is said that he seldom failed to produce a patient for this operation when requested.

Dr. Wood's work on the arterial system was enormous. It is said he tied the femoral artery over fifty times. "He ligated the carotid many times for the cure of aneurism and in one case the carotid and subclavian of the same side, and he had by this procedure successfully cured an aneurism of the arteria innominata. In the early days of his professional life he had tied the subclavian artery five times in succession and in every case cured the aneurism. . . . He tied for aneurism the external iliac eight times in succession and cured the aneurism in each case." He inaugurated the cure of aneurism by pressure.

"In surgery of the nerves he was very successful. He removed Meckel's ganglion successfully four successive times." This, too, at a period when this operation was seldom performed.

Even in those early days he performed abdominal operations, but he was prejudiced against the operation of ovariotomy, preferring to refer cases of this nature to the gynecologists. In an *in memoriam* address read before the New York Academy of Medicine, January 3, 1884, Dr. Frederick S. Dennis, who was intimately associated with Dr. Wood for many years, closed his beautiful tribute to the great surgeon (Dr. Wood died May 4, 1882) with the following words:

"Dr. Wood passed away in the unabated possession of his powers. His death was an interruption. It came to him in all the wonderful activity of his professional life, but it came as he had always expressed a wish that it should come, while he was still working. As it was he had accomplished an immense volume of work. For almost half a century he had been busily toiling for humanity . . . he always did what he could and that was much. Such a life is a lesson and an example. Fortified by the high professional achievements of Dr. Wood, this life must leave its impress upon the whole American profession."

JOHN HAMMOND BRADSHAW.

THE SURGEON'S LIBRARY

OLD MASTERPIECES IN SURGERY

ALFRED BROWN, M.D., F.A.C.S., OMAHA, NEBRASKA

THE WOUND SURGERY OF ARCÆUS

DURING the Arabian period, Spain, as the site of the Western Caliphate, passed through a period of surgical greatness second hand, so to speak, through the reflected glory of the Moorish physicians attached to the Spanish Caliph, of whom Albucasis was probably the most important. About a century after his period, in the middle of the eleventh century, the Christian reconquest of Spain began and with it interest in the arts and sciences practically ceased and very little of value came from the Iberian peninsula until toward the middle of the sixteenth century. Other countries had their wars, which were more or less of the nature of family squabbles, but in Spain the war involved people of one race and religion against people of another and consequently it was much more bitter and for the time all other things were laid aside to the end that the power of Islam be driven from Spain. The Christian reconquest is usually dated as the middle of the thirteenth century, but this date represents really only a restoration of the preponderant power and it was not until 1492 that the Alhambra fell and the last vestige of Moorish power was overcome. In the meanwhile the several smaller political entities in Spain were joining together into the two kingdoms of Castile and Aragon which were finally united in 1469 by the marriage of Ferdinand of Aragon and Isabella of Castile. Under the "Catholic Sovereigns" the country now began to advance rapidly and Spain soon became for a time the most prominent country in the world through its newly discovered colony across the seas and the wealth thus brought to it from America.

With the fall of the Alhambra in the last decade of the fifteenth century and the affluence following the discovery of America, the arts and sciences began to come into their own. Early in the sixteenth century the University at Alcalá became a noted school and medicine according to the Hippocratic doctrines was taught also at Saragossa, Valladolid, Sevilla, and other important universities, but the instruction was almost purely theoretical. There was, however, in the province of Estremadura at the Monastery of Guadaloupe, a school devoted to clinical instruction which had the special privilege of carrying on anatomical dissection, and Francisco Arceo received at least part of his instruction at this school, for he cites a case that he observed there in 1516.

Francisco Arceo was born in Fregenal in 1493. According to some authorities he obtained his education at the University of Alcalá de Henares and later went to Guadeloupe. He gained a most excellent reputation as a surgeon and attained a large practice in the country drawing patients from all parts of southwestern Europe to his residence in Llerena in the Province of Badajoz. Apparently he did no writing until late in life for his work, written at the request of the clergyman Benito Arias Montano did not appear until 1574. In the preface, dated May, 1573, Montano states that Arcæus was still alive at this time, almost eighty years of age, but possessing the same skill and manual dexterity of a man of forty. When he died is not known.

This work for the publication of which Montano and a Spanish physician, Alvarus Nonnius, are responsible is divided into two parts; one on wounds and one on fevers. The first edition was published in Antwerp in 1574 printed in Latin. It was printed in English in 1588; in German in 1614 and later; and in Dutch in 1667. The second Latin edition, the frontispiece of which is reproduced, appeared in 1658. Its title page reads "Concerning the correct method of healing wounds and two books of other precepts of that art by Franciscus Arcæus of Fregenal doctor in medicine and surgery, author. By the same, concerning the method of curing fevers. At Amsterdam. From the house of Peter vanden Berg, in the street (called) de Blauweburgwal under the sign of Mount Parnassus, in the year 1658."

The surgery does not follow the usual form of the surgeries of the period. Arcæus quotes the older authors and, differing from the majority of the writers of his time, refers to newer authors and even contemporaries, being particularly fond of John de Vigo. He then branches out from the prescribed form and, aside from taking up surgical diseases, beginning with the head and progressing to the feet, goes his own way and writes in a simple conversational style, which is most refreshing. He tells the results of his own experience and is above all else practical. In some instances, as in the treatment of penetrating wounds of the chest, he differs from the generally held opinions and plainly says so, clearly explaining his own methods of treatment. At times he digresses from the consideration of wounds and takes up other subjects. His treatment of club foot is interesting and the illustration of his home-made brace to attach to the shoe, though not handsome, looks like an efficient piece of apparatus.

Amstelodami,
ex officina Petri vanden Berge, in vico (vulgo)
de Blaeuweburgwal, sub signo montis Parnassi.
Anno 1648.

REVIEWS OF NEW BOOKS

IT is impossible to paint the lily; it is difficult even to express one's entire mental and emotional reactions to Dr. Cushing's style of expression. His thoughts are expressed simply and with a charming facility which no other writer of American surgical literature today possesses. His ability to say what he has in mind in a relatively few words and consequently in a comparatively short time must make him a godsend to the harassed arrangement committee for dedicatory exercises and like functions. The addresses in *Consecratio Medici*[1] are the result of a devotion and love for the matter in hand. It requires something more than these, however, to obtain a response from a reader who had no active part in and no relation to the exercises of the day upon which they were given.

The book consists of fourteen essays which reflect the wide range of Dr. Cushing's interests and connections. The character portrait of Samuel Garth, the Kit-Kat poet, and the chapter on "The Doctor and His Books" are evidence of the author's bibliophilic accomplishments. Perhaps unwittingly, but just as certainly, has he baited and set the trap for those young men who have had a longing for books and who, until their association with him, have dissipated their efforts. "Realignments in Greater Medicine," "The Personality of a Hospital," and "The Clinical Teacher and The Medical Curriculum" contain Dr. Cushing's views upon the question of medical education. I wonder why he has not performed the obvious experiment upon some youngster entering medicine! Certainly volunteer experimental material would be plentiful.

The volume also contains an eloquent tribute to Lister and an understanding essay upon "William Osler, the Man." It becomes more obvious each day how many characteristics the master clinical teacher and his pupil have in common. Dr. Cushing's influence upon the young men associated with him will be as great and far-reaching as was that of Osler and then, the monument they leave to medicine and surgery will be exact replicas.

Perhaps before now it will be clear that I feel that this work is indispensable to the doctor whose hobby is books.

LOYAL DAVIS.

BASED upon Sollmann's "Laboratory Guide in Pharmacology," this new work, *An Introduction to Experimental Pharmacology*,[2] has been built up with the thoroughness characteristic of its authors. The first ninety pages are devoted to chemical pharmacology, including materia medica, prescription writing, and toxicology. Experimental pharmacodynamics is dealt with in 141 pages. Appendices, covering 65 pages, are not the least valuable feature of this volume; they include lists of equipment needed, methods of administering anæsthesia to laboratory animals, different types of physiologic salt solutions, and the admirable list of doses for animals which includes the toxic and physiologically effective doses of most of the important drugs. It is thus an excellent reference work for research workers in many fields.

Many of the experiments listed are commonly performed in the courses in physiology and physiological chemistry, but the material remaining should be ample. This volume is well adapted to the needs of those who find it difficult to secure dogs for mammalian work. The experiments which the students perform upon themselves should be noted; no type of teaching is as effective as personal experience with the effects of a given drug. Explanations and discussions which should be of much value to the student accompany each section, but no attempt is made to do the student's thinking for him as is shown by the questions appended to many of the experiments.

CARL A. DRAGSTEDT.

ALL who have read the series of "Masterpieces" contributed by Alfred Brown to SURGERY, GYNECOLOGY AND OBSTETRICS will welcome this beautiful volume of historical surgical gems. It is apparent to the reader that the work has been a real joy to the author and he identifies himself among those bibliophiles who love and venerate the works of the blazers of trails.

The book consists of forty-eight sketches—one might wish them longer—of early contributions to the science and art of surgery. Some deal with well-known names; while others here and there tell of the fundamental contributions of some less well known authors. Each sketch brings to the reader the atmosphere of the ancient writer with his quaint sayings—withal a picture of the state of scientific knowledge of the time. Dr. Brown has approached each of his authors from the standpoint of contemporary history and further illuminates these worthies through citations of comparative doctrines. The atmosphere of the book is scholarly; not argumentative or doctrinal and each sketch is a model of conciseness. One may follow the browsings of the author and re-live the surgical achievements of Guillemeau, John de Vigo, Thomas Gale, Dalla Croce, Peter Lowe, and scores of others.

Himself an amateur binder of no mean skill, one might wish that Dr. Brown had added brief notes here and there on the bindings of some of the old volumes. The necessary brevity of the sketches no doubt precluded this addition.

[1] CONSECRATIO MEDICI AND OTHER PAPERS. By Harvey Cushing, M.D. Boston: Little, Brown, and Company, 1928.

[2] AN INTRODUCTION TO EXPERIMENTAL PHARMACOLOGY. By Torald Sollmann, M.D., and Paul J. Hanzlik, M.D. Philadelphia and London: W. B. Saunders Company, 1928.

[3] OLD MASTERPIECES IN SURGERY, BEING A COLLECTION OF THOUGHTS AND OBSERVATIONS ENGENDERED BY A PERUSAL OF THE WORKS OF OUR FOREBEARS IN SURGERY. By Alfred Brown, M.D. Omaha, Nebraska: Privately printed, 1928.

The temptation is strong, as one is sure Dr. Brown would wish, to secure each book described and enjoy the full the book lover rambles indulged in by the author, but alas, few medical libraries can boast the possession of many of the volumes and fewer individuals own items of so great rarity.

The reviewer cannot conclude without expressing great admiration for the typographical make-up of the volume. The title page is rubricated and is of unusual beauty. The excellent illustrations are those used in the original articles and with but few exceptions are well printed.

To congratulations to Dr. Brown should be added felicitations to the editors of SURGERY, GYNECOLOGY AND OBSTETRICS on the inauguration of this most illuminating series. IRVING S. CUTTER.

THE worker in public health should have completed a course in medicine or at least should have a great deal of information concerning medicine. But, on the other hand, a medical degree alone is not sufficient to permit the greatest efficiency in public health endeavors. One must also have a knowledge of hygiene, sanitary engineering, epidemiology, food production and handling, industrial hazards and working conditions, heating and ventilating methods, sewage disposal, and methods of collecting and evaluating statistics. In general, the physician should have a vital rôle in the field of preventive medicine and public health. It is his duty to educate the public in the ways and means of building up resistance against disease. The worker in public health, on the other hand, must be chiefly occupied with the prevention of the dissemination of disease.

In this book on *Preventive Medicine*,[1] Boyd includes a great deal of material which will be of special value to physicians, as well as others, in giving them a foundation upon which to build a knowledge of preventive medicine. The first portion deals with diseases due to microorganisms. There is first a general discussion of the sources of infection and the methods of spread. Then follows a separate consideration of each disease, with statements concerning etiology and methods of control.

Several chapters are devoted to insect borne diseases and their prevention. There is also a discussion of water supply and excreta disposal. Space is given to the deficiency diseases, occupational diseases, the puerperal state, and hereditary diseases.

The building up of individual resistance to disease is by no means the smallest part of disease control. The author of this book might be criticized for including but a small amount of material on personal hygiene and sanitation. There is no information on the hygiene of the home.

The book concludes with chapters on statistics and public health administration. It is to be hoped that the reading of this book will stimulate greater interest among physicians, educators, and the public in the prevention of disease. Those who wish to delve further into public health work will find lists of helpful references at the close of each chapter. This volume makes a good starting place.

HERMAN N. BUNDESEN.

THE new edition of Speed's *Text-Book of Fractures and Dislocations*[2] surpasses the high standard of the first edition. The text has been entirely revised and amplified to cover present day usage. The excellent first chapter on the general treatment of fractures, includes an extremely valuable section on "What to Look for in a Roentgenogram of a Long Bone Taken After Injury." The second chapter deals with the operative treatment of fractures. The third chapter is devoted to the general subject of dislocations, and the balance of the book comprises the regional discussion of fractures and dislocations. In the case of each fracture, the anatomy, pathology, and etiology are considered by way of introduction to the taking up of the symptoms, course, prognosis, treatment, complications, etc. The illustrations are abundant and include many tracings made from X-rays and photographs. While the style is admirably concise, yet the subject matter is handled in sufficient detail to furnish accurate directions to the student and physician. The multiplicity of pertinent paragraph headings facilitates the location of desired information. While the writer's wide experience qualifies him to speak authoritatively on all fractures, the section on carpal fractures possess particular merit. It is satisfying to note the writer's emphasis of the fact that "the bone lesion in skull fractures is for the most part the least significant feature; the associated damage of the cranial contents is far more important." Speed's philosophy is aptly summed up in his sentences, "Treat fractures at once with as much respect and rapidity as the acutely inflamed appendix. One should be calmly judicial in selecting methods of treatment and when a method is employed it should be made eminently efficient by thorough attention to details of technique."

FREDERICK CHRISTOPHER.

[1] PREVENTIVE MEDICINE. By Mark F. Boyd, M.D., M.S., C.P.H. 3d ed. rev. Philadelphia and London: W. B. Saunders Company, 1928.

[2] A TEXT-BOOK OF FRACTURES AND DISLOCATIONS COVERING THEIR PATHOLOGY, DIAGNOSIS AND TREATMENT. By Kellogg Speed, S.B., M.D., F.A.C.S. 2d. ed. Philadelphia: Lea and Febiger, 1928.